# THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—21ST YEAR. SYDNEY, SATURDAY, AUGUST 11, 1934. No. 6.

## Table of Contents





## THE CAUSATION AND TREATMENT OF ARTHRITIS AND ALLIED CONDITIONS.

By HORACE PERN, M.R.C.S. (England), L.R.C.P. (London),
*Leongatha, Victoria.*

### PURPOSE OF THE PAPER.

THE task I have set myself in this paper is a formidable one. I propose to discuss the subject of arthritis in all its various forms and in respect of both ætiology and the principles of treatment.

I propose first to submit to you a criticism, definitely destructive in its purport, of the report of the special committee recently appointed by the British Medical Association in Great Britain "for the promotion of research into the causes of arthritis and allied conditions", and the investigations of methods of treatment. The report of this committee was published in *The British Medical Journal* of June 17, 1933. I have adopted its title for this paper, since it allows a free field to discuss arthritis of all forms.

I propose in the second place to put before you certain observations and to deduce therefrom some general ideas regarding the essential nature of the problem with which, as practitioners, we are faced when dealing with cases of arthritis; the interpretation of these observations I am unable to give. I also wish to lay down the general principle which, I believe, should dictate our clinical attitude towards these patients.

Much of the material in this paper presents, from a different angle, ideas and methods already put forth in my previous papers on this subject.[1][2][3][4]

I propose to omit from my discussion tuberculous disease of joints, since the treatment of these is now standardized.

PART I: THE BRITISH MEDICAL ASSOCIATION REPORT.

The British Medical Association report called out some correspondence, and one letter (that of Dr. Francis Bach, *The British Medical Journal*, July 8, 1933) sets out my own views so clearly that with your permission I shall quote parts of it. He writes:

> The main object of the report of the Arthritis Committee, published in the journal of June 17th, is, in the words of your leading article, to present to the practitioner an authoritative statement on the subject of arthritis which may serve him as a guide amongst the shoals of conflicting theories and the enthusiastic assertions of the advocate of panaceas. This is a truly admirable object. It was a difficult one, and it is apparent from the report that this difficulty has been quite insurmountable. As medical practitioners in general and specialized spheres, we are seeking guidance as to the lines along which attacks against rheumatism are to be directed. Much information has been brought to us, but we are not in a position to decide what of it is essential, or immediate, or of possible later value. The present authoritative statement suggests little hope for an organized advance, but rather indicates a prospect of disorderly retreat. There is no wide survey of the situation giving indication where to advance, to entrench, or retreat.

He continues in a very thoughtful letter, of which I quote one more paragraph:

> A biased and distorted picture has been given, which, if at all understandable, is one that may well increase rather than diminish the difficulties of the practitioner.

This is strong language, but I have to say that I concur with the general impression conveyed by it.

In which ways have the committee failed where they could have thrown light?

1. Essential pathology and the interpretations deduced therefrom. I have given in the second part of my paper deductions drawn from pathology which I think conclusively prove this point.

2. Classification. The committee has built up a system of clinical entities described as distinct diseases which I believe is wholly artificial and unjustified by the facts of practical experience, at least of general practice in Australia.

3. Treatment. I should say that it was quite impossible to treat a patient with any degree of certainty by following the committee's advice, as it gives no clear indication when and how to apply rest and when and how to apply mobility. The proof of this statement is shown by the prognosis given by the committee and generally accepted.

It is, of course, impossible to traverse in any detail the findings of this important committee, or even to examine its general conclusions as embodied in the report. Nor is it necessary, for my criticism of the report concerns fundamental principles and the broad features of the problem of arthritis as a whole. I will give in abbreviation the committee's nomenclature and classification:

(*a*) Primary rheumatoid arthritis.
(*b*) Secondary rheumatoid arthritis.
(*c*) Chronic villous arthritis.
(*d*) Osteoarthritis.
(*e*) Spondylitis.
(*f*) Fibrositis.

The committee state that an ætiological classification is impossible, also a pathological one by morbid anatomy. Then they make classification more hopelessly confusing by omitting certain types of joint disease. This prevents the subject from being viewed from a whole, and thus makes it impossible to understand. Moreover, they have coined names for different pathological stages in the course of the same disease and so made confusion worse.

Long usage has left a definite impression on men's minds, so why not leave it until our knowledge enables us to classify correctly? I will deal with classification later.

### Incidence.

Some appreciation of the position can be gathered from statistics of the British National Health Insurance Scheme. Loss of work time through rheumatic diseases (of all types) totalled five and a half million days. The cost involved under the National Health Insurance Scheme, in the shape of administrative expenses and disbursements to insured people suffering from rheumatic affections (of all types) in one year is in excess of five million pounds, and for arthritic and allied conditions four and a half million pounds, and the large amount of rheumatism amongst uninsured persons adds considerably to the above figures.

### Ætiology.

Ætiology brings out nothing fresh.

1. Primary rheumatoid arthritis, cause unknown; secondary, infection.

2. Chronic villous arthritis, thyreoid deficiency and strain on joints affected due to weight are important.

3. In osteoarthritis infections makes some contribution in secondary cases, strain and arterial degeneration in primary cases.

4. Fibrositis: Many cases are due to infection, metabolic disorder and defective elimination equally common.

### Biological Chemistry.

A tremendous amount of work has been done in the biological chemistry of arthritis, but except for the blood sedimentation rate, which seems not very conclusive, no fresh light has been thrown, though it discards some old theories.

### Radiology.

Radiology throws no fresh light on the subject.

I do not propose to follow the report in detail further. I strongly recommend every general practitioner who has not read it to do so, since it contains some matter of real (that is, of practical) value. In thus "damning it with faint praise" I do not forget that it is the considered expression of opinion of men who are among the most eminent in the world in their special line of practice. But I hold it were less censurable in a medical man that he presume to stand out against some pontifical pronouncement, backed by whatever weight of authority, than that he fail to testify to the faith that is in him: "Here stand I, I can none other."

## PART II: A CONSTRUCTIVE CONTRIBUTION TO THE STUDY OF ARTHRITIS.

In this second part of my paper I propose to examine the subject of arthritis from the point of view of ætiology and essential pathology, of classification, of diagnosis, of treatment and of prognosis. In the course of this personal study I shall refer at times to the British Medical Association report.

Before I take up the subject of my discourse from the strictly clinical standpoint, I shall crave your forbearance and ask your attention while I enter upon an aspect of medicine which I believe to be particularly germane to the subject under discussion, which is not commonly included in the teaching of medical schools and which comes, not by the reading of text books, but rather by attuning one's life and outlook to an attitude of receptiveness toward Nature and the divine purpose behind Nature. To me the study of medicine is but one aspect of the varied and amazingly interesting drama of human life. The subject of disease presents itself to me, not simply as some local or even constitutional aberrant state of the tissues of the body, but rather as a divergence from the normal scheme of Nature and of physiological laws. I see in ill-health a constitutional imbalance, an inability to effect the necessary reaction to change of environment, whether these be physiological, mechanical or, shall I add, moral. In brief, disease is due to failure, inherited or acquired, to live in accordance with the fundamental laws of life.

### Arthritis and its Treatment: The Outlook of the Profession.

To understand the outlook of the profession towards arthritic conditions it is helpful to go back to the moulders of that outlook. I have obtained this information from "Treatment by Manipulation", by Timbrell Fisher.[5] John Hunter and Hugh Owen Thomas taught the power of repair in all living tissues. All have taught that the cure of inflammation is rest. John Hilton and Owen Thomas reduced rest to a science and taught that if rest was prolonged and the inflammation cured, Nature on her part would finish the cure and mobility would take place.

John Hunter taught that joint inflammation must be followed by adhesions, so when the inflammation had subsided movement was essential to prevent fixation.

Sir James Paget condemned prolonged rest; he taught that "too long rest, not only in the case of injured joints, but those kept at rest for injuries in other parts of the limb, was the most frequent cause of delayed recovery".

Sir Robert Jones taught that joints obeyed the ordinary physiological laws and that they acted mechanically; he adopted Sir James Paget's views.

So all have been unanimous that treatment of inflammation by rest is essential; they vary as to how long rest should be enforced and about the end results; they also vary definitely about the value of movements. It has been left to Sir Robert Jones to supply the needed link that joints acted mechanically, and he laid down definite guiding principles.

The British Medical Association committee, from what I can gather, follow Sir Robert, but I should say quite definitely the guiding principles they lay down are much too vague to be used as a basis of treatment.

I have been working on Sir Robert's principles; he was an orthopædic surgeon and so saw with a surgeon's eyes. I have been working on rheumatic joints; and as I am a general practitioner I look with his eyes and, being a country man, am dominated by Nature.

I have attempted in my different papers to draw a picture of the laws which govern all living tissues, so that on inspection of the joint it is possible to visualize the extent of the damage, the amount of inflammation set up to produce repair, the phagocyte at work, the formation of granulation tissue into fibrous tissue and the whole process; and because I have done that I have been accused of talking elementary physiology.

If the exquisite work of Nature may be termed elementary physiology, I humbly bow my head to it. It guides our hands during each step in treatment and dictates the only path to tread.

The general practitioner, from what I can gather, is bewildered; he has been told that rest alone will cure a joint, when he definitely knows that if rest is prolonged it must produce stiffness, in arthritic conditions most certainly. Even if the injury in the limb is away from a joint, prolonged rest will produce stiffness in it, months of treatment being needed to restore mobility. The war most conclusively proved this, as any man with experience of war work can testify. The practitioner sees every day of his life crippled joints, the result of his own failure to produce rest, of Nature's failure to produce it, and her last final effort and victory—ankylosis. He sees both rest and mobility fail, and he turns for guidance. Sir Robert Jones has given guidance in his orthopædic work.[6] The general practitioner reads the British Medical Association committee's report and, as I have already stated, the principles laid down for his guidance are too vague, the tenor of the whole report is unduly pessimistic, and naturally he is filled with despair.

### Nature and the Laws of Nature.

#### *Heredity.*

Human beings differ from all other forms of life in that they have personality. They are composed of a body, which we will call the physical body, and an individuality, which we will call the spiritual body, the two making one ego, the whole being one individuality, which is different from any other individuality.

We are immediately in difficulties with the great questions where do we come from, whither do we go, what is life, what is the object of living?

Are we just the highest form of animal life, the physical, the most important, or are we here for a purpose, each one his allotted task and the fulfilment of that task governed by the spiritual? If the latter is correct, I think it fair argument that the spiritual must govern the physical.

### *Environment.*

The ego has to live in a world or, in other words, an environment, to which it must adapt itself or fail; failure may be in the physical or spiritual or environment.

Each form of life must vary from any other form of life according to the function it has to perform and according to the environment in which it has to live; it must be capable of adaptation, otherwise it must die.

If this is all correct, we can observe Nature and the laws which govern Nature, adaptation by life to those laws, or the laws to environment in one form of life as well as another.

By common observation it is quite easy to see that certain strains or species, as, for example, rose trees and apple trees, are highly susceptible to certain forms of disease and the attack of parasites while others are immune.

I will give an example of how environment will alter that resistance. My garden is poor grey soil; my hospital garden is rich chocolate soil; they are three or four hundred yards apart. My roses seem to be immune to green fly; I never spray them, but occasionally run my finger down a shoot for a few stray flies. I can strike cuttings in my garden and when they are rooted plant them in the hospital garden (the cuttings carrying on the life of the parent tree), where they thrive and bloom about equally with my own, yet I have to spray them twice every spring for green fly.

I will quote two examples to show how change of environment is paradoxical. Violets in my garden grow in profusion, they never deteriorate, they propagate by runners, and they never seed. I can take the rooted plants to the hospital garden, where they have great difficulty in establishing themselves, and most of them die. Those that survive in a year or two go back to the small white violet; they propagate by throwing out weak runners, which usually die, but they seed in profusion, the seedlings being white violets.

Geraniums, though not to a marked extent, do the opposite. In my garden they live, but do not thrive, but they seed freely. I can take cuttings from them, and when they strike plant them in the hospital garden, where they grow in profusion but never seed.

I have given these examples and made no attempt to explain them, but simply to show in the ætiology, not only of the disease we are now considering, but in any divergence from the normal scheme of Nature and the physiological laws, that there are many problems we do not understand, and until they are solved we must of necessity work in the dark.

### Classification.

To classify the various forms of arthritis on an ætiological or pathological basis is, in my opinion, at present impossible, by reason of the fact that as yet we have not sufficient knowledge of the essential causes of the forms of arthritis of unknown or doubtful origin to make such a standard classification possible.

I will give a classification which is slightly different from that given in my previous papers. It consists of causes of arthritis of known and of unknown or doubtful origin:

(1) Traumatic.
(2) Specific infections, gonorrhœa, pyogenic, enteric, dysenteric, and in certain exanthemata.
(3) Metabolic and blood diseases, as, for example, gout, hæmophilia and purpura.
(4) Organic nervous diseases, Charcot's joints.
(5) Rheumatic fever, including its subacute stages.
(6) Rheumatoid arthritis.
(7) Osteoarthritis, which, I take it, is inflammation both of the joint and bones involving the joint.
(8) Fibrositis in all its manifestations.

All cases of arthritis, of whatever nature, can and may go through three stages, acute, subacute and chronic.

In my paper on "The Treatment of Joint Lesions of Arthritis Deformans" I divided rheumatic arthritic joints into five groups, Numbers 1 and 2 occurring during ductless gland instability, when the disease assumed an acute type, 3 and 4 during ductless gland stability, when the disease usually assumed a mild type, 5 during ductless gland deterioration, when the disease assumed a chronic type. From my own personal experience the disease we term "rheumatic fever" is very prevalent in Britain and uncommon in Australia. Is this due to environment?

### Diagnosis.

With a set of syndromes so vaguely defined as those associated under the term arthritis, diagnosis must have a different significance to that which is involved in relation to the ordinary run of diseases.

Even in respect of arthritis with a clearly defined ætiology clinical diagnosis is sometimes difficult. From the clinical standpoint the difficulty caused by lack of exact nosological classification based on ætiology is more theoretical than practical. From the point of view of treatment we can obtain our objective by merely knowing how the cause acts, whether within the joint or in the surrounding tissues, the character of the morbid changes, histological and anatomical, associated with the local conditions.

Tuberculous disease of joints may, in my experience, be extremely difficult to diagnose, though X ray specialists state that they can now identify them with a great deal of precision.

In diseases of infective origin the history usually gives the clue, and the patient's aspect immediately indicates the extent of the constitutional changes set up, whether the infection is of the virulent type, a medium or a mild one. The tongue, pulse and

temperature will corroborate your observations. Inspection of both limbs (which should always be done) tells you the locality of the hyperæmia, whether it is in the joint proper or in other structures, and the extent of that hyperæmia. It will tell you whether the condition is in a subacute or chronic stage; it will tell you what the condition of the joints is, and from the aspect of the patient you will be able to tell whether there is any ductless gland exhaustion.

Palpation gives the exact locality of the pain, and from the intensity of the pain you can judge the stage of hyperæmia; this is most valuable in diagnosis and treatment.

Mobility is the final test and, if I may be allowed to dogmatize, should never be attempted before complete relaxation is obtained. Then it should be done gently. The extent of the limitation and the amount of pain produced immediately indicate the degree of the inflammation. Both are in the same ratio. If the joint is affected, limitation will be in all or most directions. If one or more structures outside the joint proper are affected, limitation will be found in those directions which put tension on the affected structures.

The symptoms and physical signs, by their becoming less or by increase in severity, tell with a great degree of accuracy the immediate condition and progress of the case; they also give definite landmarks on which to base treatment.

To sum up, physical signs and symptoms will give clear and definite indications of the line of treatment that should be adopted, will give guidance in carrying out that treatment, and will show exactly whether progression or retrogression is taking place.

#### Pathology and the Deductions Drawn from Pathology.

I have classified the various forms of arthritis for a specific purpose, to show that entirely different causes produce arthritis, also, except for trauma, that it is a local manifestation of a constitutional disease. Inflammation is Nature's response and mode of repair. The removal of all foreign or obnoxious matter is dealt with by the reticulo-endothelial system either by fixed or wandering cells. The same process occurs in all tissues except the brain. This, I conclude, is an organized process under the guidance of the nervous system acting through the different hormones. Nature's way of assisting this is by rest, which she obtains in several ways; the most insistent one is pain. If rest is not obtained, a vicious circle is set up, leading to exhaustion of every participant in the process. That vicious circle will go on until rest is finally produced. In arthritic cases we must have a constitutional rest, both physical and mental, it being impossible to rest one without the other, also a mechanical rest for the joint or joints.

We can assist Nature, where possible, by stopping further entry of obnoxious matter, as, for example, by the removal of a septic focus or foci.

I have gone fully into pathology in all my papers and so, briefly: Rheumatoid changes occur in subacute or chronic arthritis; the subacute stage is produced either by the process of destruction going on in spite of correct rest or by the incorrect use of rest and mobility setting up another vicious circle.

The position the joint assumes is due to gravity and to the pull of the stronger group of muscles in the direction which allows the greatest room for the increase of synovial fluid. In the later stages the joint becomes permanently fixed, owing to the matting together of the surrounding tissues, which adapt themselves to their new position, either by fibrous or bony ankylosis; then rest will cure the inflammation and Nature will have gained her objective.

The duration of this process will depend on the activity of the cells, this varying with age. If patients were cured in the acute and early subacute stages, rheumatoid changes would not occur.

Osteoarthritis occurs when the physical changes begin to wane; therefore, though there is a great attempt at new bone formation, as shown by bosses of bone, atrophy goes on at a greater rate, and ankylosis does not take place.

#### Treatment.

We shall now deal with treatment. Treatment is based on the laws of all living tissues and on the physiology of joints. I have attempted to prove that rheumatoid changes are set up because those laws are broken and the joints are used mechanically in an incorrect way; therefore, if we reverse the order of things and use them correctly, we should get cure. Clinical experience bears this out.

The British Medical Association committee went thoroughly into the constitutional treatment in all its phases, therefore I shall deal entirely with the local treatment of joints. I shall attempt to give the exact reasons for the application of rest and mobility.

##### *Rest.*

The fundamental point is that, as the physiology of a joint is a mechanical one, it is absolutely impossible for it to obtain rest unless it is put mechanically at rest. If this principle, of which there can be no possible doubt, could be once grasped and taught in season and out of season, what a wonderful boon it would be to mankind.

The British Medical Association committee's report is the work of the teachers of our profession. To my reading (which, of course, may be incorrect) it clearly proves that they are not imparting the knowledge that we as general practitioners expect.

There are five questions that should be decided definitely. The historical sketch, which I have already given, shows that these questions always have been debatable and, from what I can gather, are still undecided. Under the questions would come all types of fibrositis.

1. Will rest cure inflammation? The universal answer would be: "Yes, unless the process of destruction were greater than that of construction."

2. Will rest cure inflammation of a joint? The answer would be definitely: "Yes, if properly applied and continued until all signs of inflammation had subsided."

3. After all the inflammation has subsided, will the joint obtain its full function? Most people would say: "Yes, if the period to obtain resolution has been a short one and the cause of the inflammation a minor one." But if the period of rest has been a long one and the original cause a severe one, producing much cell or tissue destruction, and so needing a lot of repair, then I am certain we can say quite definitely that adhesions will form; and, their density being in ratio to length of time and severity, these adhesions will prevent full mobility and full function.

4. Will active movements alone break down these adhesions and full functional use be obtained? The same answer may be given as to the previous question, for the same reason—it depends entirely on their density, which will be in the ratio of length of time and severity. The personal factor of the patient comes in: his courage to endure pain, perseverance and age; but we can say with certainty that if the adhesions are dense, active movements will not produce full function, or only after a long period.

5. How long should rest be continued? The answer is quite clear: Until all signs of inflammation have subsided, otherwise it will light up again. Of course, it is quite impossible to explain the degree of rest during the whole course of treatment for each individual case.

### *Position of the Joint.*

The position of the joint should be in the physiological position of rest. In the spine the normal curves should be maintained. The hip should be kept quite straight or slightly abducted as well. The knee should be in full extension. The ankle should be in full flexion and there should be slight inversion of the foot. For the shoulder the position advised is abduction; why, I know not, because in arthritis the movements lost in a shoulder are almost universally those above a right angle. Abduction is seldom lost and, if it is lost, is quite easy to obtain. The position of election should, with the arm, be straight over the head; then to obtain mobility all that would be needed would be gradually to lower the arm. (I have never tried this.) The most generally useful position is to pad the axilla, sling the hand close under the chin and fix the arm to the side. The elbow should be kept at about a right angle, and the wrist in full extension.

### *Mode of Application.*

We can assist the reticulo-endothelial system by preventing destruction of immature cells, which we have already done by rest, limiting the outpouring of exudate, assisting drainage by maintaining a uniform pressure, a uniform temperature and, lastly (I shall mention this under "Mobility"), by obtaining full functional use.

If these principles were adhered to and if resolution were our only objective, any mode of application would do. But as there are equally important factors to be taken into consideration the mode of application becomes of great importance.

Pyogenic affections which may destroy either the life or limb call for absolute rest with some form of splint, with extension if required. In all severe traumatic cases primary union and stability of the joint are the main essentials; thus absolute rest is required. The minor traumatic conditions, most of the cases of arthritis of known origin, and, I am perfectly certain, those of unknown origin, do not require absolute rest. I consider that for these cases wool pressure is ideal. I have given details of its mode of application several times. The amount of wool must be abundant—according to the thickness of the wool, three or four complete turns round the limb. The wool should extend well above and below the joint, the bandage should be of firm substance and should be applied firmly.

For mild injuries in structures outside a joint, firm strapping, either to support the structures or to limit movements, is all that is needed.

Whatever mode has been adopted, except in those cases in which treatment by strapping alone has been chosen, the joint finally should be firmly strapped to give support when it assumes its normal function. All traumatic conditions are treated on general surgical and mechanical principles.

### *Mobility.*

I think we can accept as an axiom that all life, and each cell which helps to form each individual, must perform its own function or deteriorate. The tissues of the trunk and extremities are for a mechanical use and their health depends on correct and sufficient use to maintain their normal tone; failure of one or the other will cause deterioration. This is well seen in chronic joint affections when all structures, including the bones, show signs of degeneration.

The problems we have to consider when dealing with limitation of movement in one individual or many joints are:

1. The effect on the whole ego—how much that limitation will alter that ego's adaptation to the environment.

2. As a joint is the fulcrum of a system of levers, how much each fulcrum will alter the whole system of levers affected.

3. The effect on each individual joint.

I have already dealt with Number 1. Number 2 I dealt with in my last paper on the treatment of *arthritis deformans*; and I pointed out that the function of the upper extremity comprised the numerous uses to which it was put, and that if one joint was affected it might profoundly alter the complex functions performed by the whole

extremity. The function of the lower extremity is to bear the weight of the body and to subserve locomotion; if the centre of gravity is altered in one fulcrum it must affect the whole, and the affection of one joint the whole of locomotion. In dealing with subacute and chronic multiple arthritis, this is very much brought home and is one of the main difficulties to be overcome; it is also the cause of much disappointment. Number 3 we shall deal with as we go along.

*Causes of Limitation of Movement.*

The causes of limitation of movement are as follows:

1. In the acute stage muscular spasm is set up to produce rest and to protect highly vascular tissues and to allow the process of repair to go on.

2. At the termination of the acute stage and when complete subsidence of inflammation occurs, adhesions of varying density are left.

3. In the early part of the subacute stage spasm of muscles occurs to guard the highly vascular joint structures, and surrounding tissues are infiltrated. In later stages the joint assumes a fixed position and is held there as a result of alteration in the structures which surround the joint adapting themselves to their new position.

4. In the chronic stage, which may be a continuation of the subacute stage, or which may occur when all the inflammation has subsided, a contracted capsule is left and there occur partial or complete destruction of the cartilage and dense fibrous or bony nodules or bony ankylosis.

*Modes of Obtaining Mobility.*

Mobility may be obtained: (i) by active movement, which is performed by the patient unaided; (ii) by passive movement. Passive movement may be applied in five ways: (*a*) By application of a firm support of the limb to overcome spasm of the muscle and to allow the patient to perform active movement. (*b*) By gravity, the weight of the limb performing the movement. (*c*) By altering the position of the limb at certain intervals. (*d*) By passive movement performed by the operator. This movement can be done daily or at frequent intervals gradually to overcome the cause of the limitation, gradually fretting away the obstacles and producing only that degree of trauma that the joint structures are capable of dealing with, without setting up fresh inflammation. (*e*) By forcibly, at one or more sittings, breaking down the causes that limit mobility.

*Acute Stage.*—If complete resolution takes place in a short period, the joint will have full mobility and no further treatment is required, though after all causes active movements will strengthen all the structures participating in the use of a joint. These movements should always be carefully explained and the movements should be the full range of the joint, and they should be done slowly and deliberately. If the acute stage is prolonged and inflammation continues in spite of rest, adhesions will form, and we have to devise a treatment which on the one hand will not destroy highly vascular cells and so prolong the inflammation, and on the other to prevent the formation of adhesions. This can be done by passive movements; active movements unaided must do harm and should never be allowed. Passive movements should be commenced early and should be of the type of modified passive movements such as I have already described. Alteration of position can be carried out with perfect safety as long as reasonable care is used. Assisted passive movement requires practice. Complete relaxation is most essential; the limb should be held firmly and the ends of the bone should be gently pulled apart, the operator regulating the pressure he exerts so as not to impede the movement. The extent of the movement is the full range allowed and (this point I wish very much to stress) no pain, or only very slight pain, should be produced, the movement should be done once, and once only, the joint then being put at rest.

If the operator has sufficient experience, he can give passive movements unaided with perfect safety. I have been doing this for years. When resolution finally takes place, with or without limitation of movement, active movements are quite safe and should be ordered.

In regard to subsidence of the inflammation which leaves adhesions of varying density, if I read the writing on the wall correctly, manipulation is going to be the vogue; and it fills my heart with dread, for I think we shall return to the days of Thomas and the abuse of manipulation; another Thomas will arise to preach rest, and rest only, and we shall have no progress. So I want to bring this out so clearly that there can be no misinterpretation: if the adhesions are likely to be fine, then manipulation at one sitting, with or without an anæsthetic, is not only safe but is dramatic. The extent of the movement must be the full range of all the movements of the joint (compared, if necessary, with the joint of the opposite side) and one movement is sufficient. The full range of active movement should be explained to the patient and performed as soon as possible.

If the adhesions are dense, the different modes of breaking them down are: (*a*) Passive movements carried out daily or at frequent intervals, gradually fretting away the obstructions and producing only the trauma that the joint can deal with without setting up clinically recognizable inflammation; pressure, this is applied at intervals. (*b*) Pressure can be applied continually by some mechanical appliance (of this I have little experience). From years of experience of the former I am certain that this is the correct method, and all joints will with time and patience recover, except those in which the condition has progressed so far that passive movement becomes a mechanical impossibility. (*c*) At one sitting or at intervals to break down adhesions partially under an anæsthetic. I have no experience of this mode, but in highly expert hands I should think it would be quite safe, as long as only a very mild form of inflammation was set up,

which could be dealt with by passive movement and after the subsidence of the inflammation by active movements. (*d*) Forcible movement at one sitting. One cannot too clearly understand that the weakest structure must give way, whether that is an adhesion, a structure involving the joint proper, or a structure connected with the use of the joint; this may permanently disable the joint and, according to the extent of the trauma, inflammation will be produced. If the joint is put completely at rest to cure the inflammation, the adhesions will immediately reunite and become denser than ever. If active movements are ordered, they will be impossible owing to spasm of the muscles, and if they are attempted, they will cause further inflammation. Passive movement in one of its modifications may save the day, but it must be given in the same way as for an acutely inflamed joint. (*e*) Active movements alone I have already dealt with. The point to remember is that pain must be produced if there is to be any progress. Pain can be completely ignored if it subsides quickly, but if it is prolonged then it is a sign of recurrence of inflammation. If this or any other sign of inflammation should occur, active movements must be stopped immediately. This should be explained to the patient most carefully.

*Subacute Stage.*—In the early subacute stage, which is simply a prolongation of the acute stage, the same treatment that I have already described is applicable, that is, rest and mobility.

In the late subacute stage and in the chronic stage where inflammation is still present, the joint has assumed a fixed position. These stages should never occur, but still are in most cases curable with rest, with passive movements to overcome the obstruction gradually, and with active movements to consolidate each new position. I think forcible manipulation to break down suddenly the obstruction which limits mobility is contraindicated and can only lead to disaster. I went fully into the treatment of each joint in my last paper on "The Treatment of Arthritis Deformans".

*Chronic Stage.*—In the chronic stage and when inflammation has subsided, rest is not needed; the treatment is passive and active movement.

To sum up, the passive movements which I have most earnestly tried to explain need a trained hand that can gauge pressure to the finest degree, the power to modify that pressure from the finest delicacy of touch to all the force the operator can exert, the ability to move a joint so that no sign of inflammation is set up, to heal and not to destroy. It needs endless patience and there are many disappointments, but it does bring its own reward of being able to watch a crippled joint gain its function again and of seeing somebody who had given up hope able to take up life again and face the future.

### Prognosis.

You all have your own personal experience, the British Medical Association committee's report, text books, and current literature, and have no doubt come to your own conclusion.

### Conclusion.

I have attempted in my paper to show that though the road may wind up hill all the way and "the journey take the whole long day", the night will pass and with it its fears and dreads. The dawn will break and with the dawn the mists will melt and full day will bring understanding, and with it crippled joints and broken lives will belong to the night that is passed. This conviction has compelled me to write these papers.

### Acknowledgement.

I desire to express my sincere thanks to a friend, who wishes to remain anonymous, for his most helpful suggestions in the compilation of this paper.

### References.

(1) Horace Pern: "Common Joint Conditions", THE MEDICAL JOURNAL OF AUSTRALIA, August 6, 1927, page 176.
(2) Horace Pern: "Rheumatoid Arthritis", THE MEDICAL JOURNAL OF AUSTRALIA, July 13, 1929, page 44.
(3) Horace Pern: "Arthritis Deformans", THE MEDICAL JOURNAL OF AUSTRALIA, August 6, 1932, page 169.
(4) Horace Pern: "The Treatment of the Joint Lesions of Arthritis Deformans", THE MEDICAL JOURNAL OF AUSTRALIA, May 13, 1933, page 573.
(5) Timbrell Fisher: "Treatment by Manipulation."
(6) Sir Robert Jones: "Orthopædic Surgery of Injuries", Volume I.

## PARALYTIC ILEUS.[1]

By KEITH ROSS, M.S.,
*Geelong, Victoria.*

IT is with misgiving that I submit this paper, for the subject of it is, as Sampson Handley(1) so truly remarked, "surrounded by that indefinable haze, the result of imperfect knowledge, which to the despair of the student envelopes certain chapters of surgery". It is not for me to attempt the Herculean task of clearing this haze, but rather to redirect your attention to the fact that it still remains, despite an interval of twenty years, almost as dense as ever.

Paralytic ileus has been defined as a condition in which, in the absence of mechanical obstruction, the intestine becomes progressively distended with fluid and gas and loses its power of contraction. It has varying grades of severity and may affect various parts and lengths of the intestine.

It is seen most frequently in association with peritonitis, mechanical obstruction, after abdominal and other operations, after severe colic, after injuries to the abdomen and chest, after embarrassment of the intestinal circulation, and also as a complication of severe and debilitating diseases. But, however wide we make the ætiological factors, there remains a residuum of cases in which it is difficult to ascribe a cause.

### The Mechanism of Ileus.

The mechanism of paralytic ileus is obscure. We know that the bowel is a muscular organ, within the walls of which lies a local nerve plexus, that this plexus receives impulses from the *medulla*

[1] Read at a meeting of the Victorian Branch of the British Medical Association on May 12, 1934.

*oblongata* through the vagi, and from the fifth to the eleventh thoracic segments of the spinal cord through the splanchnics. According to Samson Wright,[2] movements of the small intestine are of two kinds—peristalsis and segmentation. Normal peristalsis, he says, is probably due to a series of coordinated local nervous reflexes in response to the chemical and mechanical stimulation set up by the food, while segmentation may be the response of smooth muscle to distension. Stimulation of the vagi increases both the tone and the movements of the intestine, while stimulation of the splanchnics diminishes them. This is as far as our knowledge goes, and we are completely in the dark as to just why and how tone is lost and movements cease. Possibly different factors operate in different cases. Thus it may be that the intestinal muscle itself is injured, or Auerbach's plexus, or that inhibitory impulses are carried by the extrinsic nerves.

Whatever may be the mechanism, the clinical aspects of this condition are familiar to us all. In its minor manifestations it may appear as a mild and temporary abdominal distension; in its most severe form the abdomen is hugely distended, vomiting is usually frequent and effortless, constipation is absolute, and the patient lies with drawn, sunken features, cold extremities, clear eyes and alert mind, in imminent danger of death.

### Diagnosis.

As a rule diagnosis is easy, but at times there may be much difficulty in differentiating it from: (*a*) mechanical obstruction, (*b*) acute dilatation of the stomach, and (*c*) peritonitis. With regard to the two first mentioned, a careful evaluation of the early symptoms and also the physical signs will usually give the correct diagnosis. Peritonitis is a different proposition, for it is one of the most common causes of paralytic ileus. Frequently the position is that a decision has to be made as to whether the patient is suffering from ileus only or ileus *plus* peritonitis. The general history of the case and the temperature may be of help, but personally I know of no infallible way of always arriving at a correct diagnosis.

Special attention may perhaps be drawn to crush fractures of the lower ribs, for severe ileus is not uncommon after such injuries, and here one is faced with the problem as to whether the distension is wholly due to the chest injury or whether there is an associated intraabdominal lesion. Close observation for a few hours frequently gives the clue.

### The Cause of Death in Ileus.

Before proceeding to treatment we must first consider the cause of death in ileus, and here we are brought face to face with the most profound of all the many mysteries of the subject, a mystery which causes ceaseless controversy amongst those who seek to unravel it. It appears that there are two main schools of thought. The first holds that death in obstruction is due to loss of chlorides and water; the second maintains that while dehydration and alkalosis may be the main factor in simple obstruction, or at any rate in its early stages, there is a second factor, toxæmia, which is of supreme importance in many mechanical obstructions. Amongst the supporters of this second view there is, despite much devoted research, no agreement either as to the nature of the toxin, its origin, or even its mode of absorption. It has been claimed that the toxin is a proteose, an amine, a nucleo-protein, an alkaloid, a ptomaine *et cetera*, that it has its origin from the food, the stomach, the duodenum, the pancreatic secretions, the intestinal mucosa, bacteria *et cetera*, that it is absorbed via the blood vessels, the lymphatic vessels, the peritoneum. It is impossible in a paper of this nature to thread a way through the maze of apparently contradictory experiments, and our purpose may best be served by noting the conclusions of Cooper,[3] who has made a masterly and critical review of the literature. He writes:

> Certain facts stand out and should be emphasized.
>
> 1. In high obstruction two different processes are involved: (*a*) a profound disturbance in the acid-base mechanism resulting in alkalosis and dehydration, and (*b*) a definite toxæmia.
> 2. There is a toxin in the lumen of the obstructed gut and it looks very much as if it reached the blood stream and was responsible for the toxæmia. The origin of this is not clear.
> 3. There seems to be some mysterious connexion between the toxæmia of high obstruction, acute pancreatitis, bilateral suprarenalectomy and anaphylaxis.
>
> What is the cause of death in high obstruction?

Cooper's paper was published in 1928, and since then opinion has tended rather to veer away from toxæmia as a factor; however, it is doubtful whether finality has yet been reached.

### Treatment.

In the light of the foregoing it might at first sight appear hopeless to discuss treatment. Still, the laboratories have given us some, if few, facts, and clinical experience supplements these with what may best be termed impressions. Thus treatment need not be wholly unenlightened. For the sake of clarity various common forms of treatment will be set out and discussed separately.

#### *Prophylactic Treatment.*

While prophylactic treatment is of supreme importance, it is now so generally known that it need be given only the briefest mention here. The days are gone when patients were purged, starved and generally maltreated prior to operation. Also we all appreciate the value of gentle handling of tissues exposed at operation. Post-operative treatments still differ considerably, but it is generally conceded that a certain amount of rest is due to both the patient and his bowels. My own routine post-operative treatment is to give a glycerine enema every second day for the first week.

#### *Heroic Sedative Treatment.*

Some surgeons adopt what I have termed heroic sedative treatment either as a routine measure or in the presence of distension. Morphine is given

until the respirations fall to the region of twelve per minute, and no effort is made to open the bowels. We are accustomed to think that morphine causes constipation, but a certain amount of experimental evidence is accumulating that it may increase peristalsis and tone. However that may be, I have never used this treatment myself, though I have had the opportunity of observing its results.

*Heroic Stimulant Treatment.*

Heroic stimulant treatment is the very opposite of heroic sedative treatment, every effort being made to get the bowels open early and often. Purgatives, enemata and hypodermic injections of various drugs may all be brought into action. About ten years ago a number of papers were published extolling the virtues of repeated injections of pituitrin and eserine in commencing ileus. It was at this time that I had the following experience.

CASE I. A robust young man was admitted to hospital with a perforated duodenal ulcer of eight hours' duration. Closure was simple, but some distension of the intestine was noted. Stimulant treatment was instituted, that is to say, small doses of pituitrin and eserine were given every alternate hour, also a purgative every evening, and if the bowels did not act, as they did not, an enema was given every morning. Death occurred on the sixth day. An autopsy disclosed nothing untoward but grossly distended intestine.

Never since then have I used heroic stimulant treatment, but I have observed it in the hands of others and have seen no reason to alter the opinion that it is the worst possible form of treatment. I have spoken of it at more length than it deserves, for unfortunately it does make its appeal to those who feel that with distension increasing they must be doing more and more.

*Comparative Sedative Treatment.*

By comparative sedative treatment is meant a blending of the above two forms with a leaning to the sedative side. Morphine or other sedatives are given to insure the patient rest at night and reasonable freedom from pain by day. Infrequent efforts are made to open the bowels. While many surgeons like a "good dose of oil" or other purgatives, I prefer enemata. In my experience nothing is so effective in evacuating flatus as a turpentine enema given twenty minutes after a hypodermic injection of one cubic centimetre of pituitrin. These may be repeated at intervals of twenty-four hours, but longer if possible, as too frequent enemata may cause more distension than they relieve. The rectal tube does no harm and occasionally a surprising amount of good. Oral administration of fluids is restricted or withheld, hot bags or stupes are applied to the abdomen, and the patient is made as comfortable as possible in both body and mind. This is the treatment to which I pin my faith.

*Gas Gangrene Serum.*

In 1926 Williams[4] expounded the hypothesis that the toxæmia of intestinal obstruction was due in part at least to *Bacillus welchii* toxin. A good case was made out and for a time commercial laboratories had more difficulty in supplying than in disposing of their serum. It is doubtful if the original high hopes have been fulfilled, but as the serum still has many advocates among practitioners generally, it may be worth while to examine briefly its claims. To those who believe that the toxin of obstruction has a bacterial origin, Cooper puts the following queries:

1. Why is it that high obstruction is so much more fatal than low, when the low small intestine contains so many more bacteria than the upper?

2. Why does the toxin appear so rapidly after obstruction? Its appearance has been reported three hours after duodenal obstruction.

3. Why does the toxin appear with such rapidity in the intestine of dogs that have received injections of it? It has been repeatedly shown that the intestinal contents of an obstructed loop of gut, or a closed loop, or a strangulated loop, after being prepared in any of a number of ways and injected intravenously into another animal, will cause the rapid death of the second animal with symptoms of a profound toxæmia. Furthermore, the intestinal content of an animal that has so died from toxic injection is also toxic when used in a similar way, and this process can be repeated indefinitely, the toxin always being recoverable from the intestinal content of an animal that has died from its injection.

4. Why does the toxin appear in cases in which there is no reason to suspect stagnation of intestinal contents? It is reported in acute pancreatitis, thrombosis of the portal vein, bilateral suprarenalectomy, and distemper.

Finally, reverting to *Bacillus welchii* in particular, it should be noted that *Bacillus welchii* toxin is readily destroyed by heat, whereas the toxin universally conceded to be present in obstructed intestinal contents is not affected by boiling. Thus it would appear certain that the toxæmia of intestinal obstruction cannot be ascribed to the *Bacillus welchii*. However, I am unaware of any experiments that have conclusively proved that *Bacillus welchii* antitoxin is of no avail in combating the toxæmia and promoting peristalsis. Williams's own experiments suggest that it has some such power. Furthermore, the opinion of nurses, who, in my experience, are unanimous that the antitoxin is beneficial, cannot be entirely disregarded.

In ileus associated with peritonitis we may be on surer ground, and my own practice is to employ gas gangrene serum as a precautionary measure in infective conditions such as perforated appendices or where there has been soiling of the peritoneum.

*Sodium Chloride.*

For many years the value of combating dehydration by administration of fluid has been appreciated, and while various percentages of various substances were given with the fluid, the solution in most general use was normal saline, that is, a 0·9% solution of sodium chloride. It appears that we were entertaining an angel unawares, for it is only within the last ten years that the virtue of sodium

chloride *per se* in intestinal obstruction has become known. It was then first recognized that the blood chlorides in obstruction are definitely lowered, as large amounts of chloride are lost in the vomitus or in the quantities of fluid filling the dilated intestine. Some authorities hold that it is necessary to administer chloride merely to replace that lost; others think that chloride has a direct detoxifying effect. Whatever may be the process involved, it is generally agreed that it is only the sodium salt that is so beneficial. This necessity for sodium may be due to the fact that sodium forms 90% of the base of the body.

Having decided that it is essential to give sodium chloride, there remains the practical point of how it is best administered. It is wise to remember that the patient needs water as well as salt. Rectal injection is frequently all that is needed, and if a satisfactory continuous apparatus is employed, many patients can absorb five or six pints of normal saline solution in twenty-four hours. Subcutaneous injections are most commonly made under the breasts, and possibly there is less risk of sloughing if normal saline solution is used in preference to more concentrated solutions. When saline is given intravenously, normal, double strength or 2% solution is most frequently employed.

The following case illustrates the dramatic results that sometimes follow chloride administration.

CASE II. A posterior gastro-jejunostomy was performed on an elderly and very debilitated man who had a massive indurated ulcer at the pylorus. Within a few hours the patient commenced to return his rectally administered saline solution and distension was noted commencing in the lower part of the abdomen. Severe ileus rapidly ensued and would not respond to ordinary treatment. Finally, on the third night after operation, 1,200 cubic centimetres (two pints) of double strength saline solution were given intravenously and the stomach was washed out by giving repeated drinks of sodium bicarbonate solution. By the next morning the bowels had acted twice of their own accord and, when visited, the patient was placidly reading the paper. An uninterrupted recovery followed.

American surgeons have for some time been advocating continuous intravenous medication for this and other conditions, and one has wondered how surgeons there have persuaded their patients to keep still for so long. Bailey and Curnow[5] have recently answered this conundrum and advise using a vein in the leg to which a Thomas splint has been applied.

### *Jejunostomy.*

Jejunostomy and ileostomy, particularly the former, are in high favour in many quarters, and are used both as prophylactics and cures. It is claimed that if, when operating in a case of, say, mechanical obstruction with gross dilatation of the proximal part of the gut, or peritonitis with abdominal distension, one, after attending to the primary conditions, sews a tube into the jejunum, one has insured against ileus. At one time I subscribed to this view and performed prophylactic jejunostomy some half dozen times with satisfactory final results. The following case made me wonder.

CASE III. An extensive irreducible intussusception of the jejunum was resected from a man of thirty-six. End-to-end anastomosis was performed and also jejunostomy a few inches above the suture line. Severe ileus ensued and there was very little drainage from the jejunostomy. Three days later ileostomy was performed. During the next two days the total drainage from both tubes was under two pints and the patient remained *in extremis*. On the following day a satisfactory result was obtained from an enema, and both tubes then began to drain profusely.

The conclusion could not be escaped that the safety vents functioned only when the need for them had passed. Shortly afterwards a somewhat similar case yielded an identical experience. Since then I have never performed either jejunostomy or ileostomy for paralytic ileus, except in one instance, when I wished to convince some unbelieving house surgeons of the uselessness of this procedure. Luckily the demonstration was successful.

Another benefit claimed for jejunostomy is that fluid can thus be administered directly into the bowel. Although the wisdom of attempting this is doubtful, my experience has been that extremely little of the fluid injected into paralysed gut has been retained.

Jejunostomy undoubtedly has its uses, one of which may possibly be found in the prophylactic treatment of ileus, but in the treatment of developed ileus my experience, small though it is, makes me suspect that its reputation may be based on inaccurate observations.

If properly performed, a jejunostomy will readily heal, thus it may be argued that it is reasonable practice to do it on the grounds that even if its benefits are problematical, at least it is unlikely to do harm. Some surgeons do claim that it may do harm by diverting essential substances from the lower part of the bowel, but, apart from that, the view must be put that any treatment that does not have a definite prospect of doing good is unwise, for a multiplicity of efforts is apt to detract the attention from more essential treatment and also to engender a false sense of satisfaction with our efforts.

### *Ileo-Colostomy Plus Caecostomy.*

Handley, in his famous paper on *ileus duplex*, devoted attention to cases of pelvic peritonitis causing ileus. He demonstrated that the loops of bowel in the pelvis were inflamed and collapsed and that those above the pelvis were distended. For this condition he advocated ileo-colostomy *plus* caecostomy. I am not competent to discuss the merits of this treatment, for I have never practised it myself or seen it practised. Caecostomy alone has also been advocated for similar cases. I have never seen this done, but my first teacher in surgery taught me to regard a caecal fistula following operation for severe appendicitis as a happy omen.

### *Mechanical Drainage.*

Certain methods of emptying the intestines have occasionally been advocated, the claim being made that when operating on mechanical obstruction it is beneficial to empty the dilated proximal gut. To my knowledge these methods have not found very

general favour, and I have had experience of only one case and in it drainage occurred by accident. As the case was one in which paralytic ileus would not have been surprising, it may be worth quoting.

CASE IV. A woman of sixty, a chronic invalid, had an acute obstruction of three days' duration. Obstruction was due to a volvulus of the small intestine, the twist having occurred around adhesions to the right broad ligament, the site of a previous operation. In order to divide the adhesions, the bowel had to be eviscerated. It was tremendously dilated and œdematous, and as it hung over the patient's side it ruptured, luckily in a dependent loop. By gentle pressure several pints of intestinal contents were evacuated before the rent was sutured. Saline solution and *Bacillus welchii* antitoxin were administered and there was surprisingly little post-operative distension.

*Stomach Tube Drainage.*

Those of us who were brought up in the bad old days of the large stomach tube will, I fancy, still have cause to remember it. As a house surgeon I was ordered to employ it on three occasions for paralytic ileus. Although both the patient and his attendant were in each instance reduced practically to extremis, it is only fair to record that all finally recovered. Whether this was due to any beneficent action of the tube, or whether it was that the patients then realized how dreadful death could be, I cannot say.

If our object is merely to wash out the stomach, repeated drinks of sodium bicarbonate solution will usually have the desired effect without causing undue distress. If, on the other hand, we wish to decompress the intestine by drainage, suction can be applied to a small stomach tube. This is being strongly advocated by some American surgeons at the present time. Gastrostomy has also been advised for the same purpose.

*Administration of Bile.*

Brockman[6] claimed that the rectal injection of bile was a prime factor in the very good results he obtained in obstruction. Confirmatory opinions are slow in forthcoming. Experimentally, Benedict and others[7] arrived at no very definite conclusions. There is, of course, some difficulty in obtaining human bile when it is needed.

*Acetylcholine.*

Acetylcholine has recently been enthusiastically advocated by Abel.[8] I have had occasion since reading his paper to use it only once, when it appeared to have an effect, beneficial, but less dramatic than in the cases quoted by Abel.

*Spinal Anæsthesia.*

Following on the observation that spinal anæsthesia sometimes induced contraction of the intestines with a free evacuation of fæces, it was suggested a few years ago that here was a remedy for paralytic ileus. Apparently it has had some success, for it is still being advocated. However, it is to be noted that objections have been raised on the grounds that it is unwise further to lower the blood pressure in these debilitated patients, that if a satisfactory contraction does occur, the upper intestines may be flooded as well as the lower, and also that in peritonitis infection may be spread by the contraction. My own experience is limited to one case, as follows:

CASE V. Caecostomy was performed on a man of fifty-eight, who had acute obstruction due to a carcinoma of the splenic flexure of the colon, and a large quantity of fluid material was immediately evacuated. Despite chlorides and other treatment the patient's abdomen became progressively more distended and drainage from the caecostomy ceased, although its patency and that of the ileo-caecal valve were verified by digital examination. On the third day spinal anæsthesia was induced to the level of the nipples. There was no evacuation of fæces or gas, and death occurred on the following day. Autopsy revealed no cause of death other than paralytic ileus.

**Conclusion.**

This concludes our survey. It is one which is sketchy and full of gaps, and no one is more conscious of this than I am. Nevertheless an attempt has been made to fill in the broad outlines. In the realm of treatment I have ventured from time to time to give my own views, and I have done so, not because I have any claims to knowledge, but because each of us, as he goes along, has his own experience and slowly and painfully builds up his own faith; and a paper of this nature should, I think, reveal whatever faith is in its writer, even though that faith be misplaced.

**References.**

(1) W. Sampson Handley: "Ileus Duplex", *The British Journal of Surgery*, Volume III, 1915-1916, page 161.
(2) Samson Wright: "Applied Physiology", Fourth Edition, page 432.
(3) H. S. F. Cooper: "The Cause of Death in High Obstruction", *Archives of Surgery*, Volume XVII, 1928, page 918.
(4) B. W. Williams: "The Importance of Toxæmia due to Anaerobic Organisms in Intestinal Obstruction and Peritonitis", *The British Journal of Surgery*, Volume XIV, 1926-1927, page 295.
(5) Hamilton Bailey and J. M. Carnow: "Continuous Intravenous Saline", *The British Medical Journal*, January 6, 1934, page 11.
(6) R. St. L. Brockman: "Arris and Gale Lecture on the Toxæmia of Acute Intestinal Obstruction", *The Lancet*, Volume II, 1927, page 317.
(7) E. B. Benedict, C. P. Stewart, P. N. Cutner: "The Rôle of Bile in High Intestinal Obstruction", *Surgery, Gynecology and Obstetrics*, Volume LIV, 1932, page 605.
(8) A. L. Abel: "Acetylcholine in Paralytic Ileus", *The Lancet*, December 2, 1933, page 1247.

## THE BACTERIAL FLORA OF THE MOUTHS OF AUSTRALIAN VENOMOUS SNAKES IN CAPTIVITY.

By F. ELEANOR WILLIAMS, MAVIS FREEMAN, B.Sc., and EILEEN KENNEDY, B.Sc.
(*From the Walter and Eliza Hall Institute, Melbourne.*)

SUBCUTANEOUS abscesses have not been infrequent during the process of immunizing animals against Australian and other snake venoms. This fact, together with the finding by Kellaway and Williams[1] of anaerobes in an unfiltered commercial preparation containing snake venom from another country led us to investigate the bacterial contamination of venom. We have studied the bacterial flora of the mouths of snakes, both freshly captured and after some time in captivity, and the bacterial content of freshly collected venom and of venom after drying for various times.

During the period from April 26 to November 8, 1933, we examined the mouths of 49 snakes, of

which 16 were freshly caught and the remainder had been in captivity at the Melbourne Zoological Gardens for some months. Of these 49 snakes, 21 were tiger snakes (5 freshly caught), 4 black tiger snakes (in captivity over two years), 17 copperheads (4 freshly caught), 3 brown snakes (all freshly caught), and 4 black snakes (all freshly caught).

This portion of the investigation, except the observations upon freshly caught snakes, was carried out at the reptile house at the Melbourne Zoological Gardens. After the snakes had been "milked" in the routine manner by allowing them to bite through rubber dam stretched across small medicine glasses, the snakes' mouths were held open with forceps and swabs were taken. Smears and cultures into alkaline meat medium were made immediately. The examination of freshly caught snakes and of the venoms was made at the Walter and Eliza Hall Institute.

The venoms of 38 of these snakes and those from a further 12 snakes (11 tiger snakes and 1 copperhead) were also examined both immediately by smears and culture and after drying for various periods by culture into alkaline meat medium. No attempt was made to sterilize the vessels in which venom was collected. As for routine collection of venom, the vessels were washed and mechanically cleaned and the rubber dam was washed in spirit before being stretched over their mouths.

### Examination of Direct Smears.

Smears stained by Gram's method from the mouths of snakes which had been in captivity contained multitudes of organisms—Gram-negative bacilli, Gram-positive bacilli and cocci, large Gram-positive rods resembling anaerobes, spirochætes (which we were unable to culture or to study further), and in one copperhead, numerous protozoa. The films from snakes with "cancre" and from those with apparently healthy mouths were indistinguishable.

In contrast, smears from the mouths of freshly caught reptiles showed no organisms except a few Gram-negative bacilli in the smears from one tiger snake and a few mixed organisms in those from the black snakes. The smear from one black snake showed also a few organisms resembling spirochætes.

The smears from the freshly collected venoms showed no organisms, except in five snakes in captivity, in which a few were observed, thus demonstrating the efficiency of the method for collecting venom uncontaminated by saliva.

### The Bacterial Flora of Snakes' Mouths.

#### *Coliform Bacilli.*

One or more species of coliform bacilli are frequent normal inhabitants of snakes' mouths. A large number of these organisms, almost all differing from each other in their fermentation reactions, were isolated from the mouths of 73% of snakes studied. We failed to isolate them from one copperhead, three black tiger snakes, seven tiger snakes, and two black snakes. Of these, only the two black snakes were freshly caught.

#### *Non-Lactose-Fermenting Gram-Negative Bacilli.*

One or other of two strains of *Bacillus proteus* was frequently isolated from the mouths of snakes in captivity, but not from freshly caught snakes. Both these strains are motile, liquefy gelatine, digest milk and blood serum, give a positive indol reaction, have a putrefactive odour, and give a spreading growth with the development of a brownish colour on agar. One of these ferments mannite, and the other salicin, with the production of acid and gas. Both ferment dextrose, saccharose, maltose, galactose, and lævulose, with the production of acid and gas. *Bacillus proteus* was isolated from six out of seven of the copperheads and from four out of six of the tiger snakes with "cancre" which were cultured. It was never found in any freshly caught snake and was not isolated from any tiger snake without "cancre", but was found in five copperheads without "cancre" and in one which had a discharge from the nares. It was also isolated from two of four black tiger snakes which had been two years in captivity. One or other of these strains was found in 35% of the mouths of the snakes examined, and more than half the snakes in which *Bacillus proteus* was present had obvious "cancre".

From two of the black tiger snakes *Bacillus pyocyaneus* was isolated, and from one of the copperheads a strain of *Bacillus fæcalis alkaligenes*. Many other strains of non-lactose-fermenting organisms were isolated, one or more being present in all the snakes investigated. The fermentation reactions of a few of these are set out in Table I.

TABLE I.

*Non-Lactose Fermenting Organisms.*

| Number of Organism. | Motility. | Mannite. | Salicin. | Dextrose. | Saccharose. | Maltose. | Galactose. | Lævulose. | Indol. | Methyl Red. | Agglutination against Anti-Serum made for Organism Number 1. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | + | – | – | AG | AG | AG | AG | AG | + | + | 1/800 |
| 2 | + | – | – | AG | – | AG | AG | AG | + | + | – |
| 3 | + | AG | – | AG | – | AG | AG | AG | – | + | – |
| 4 | + | – | – | A | A | A | A | A | + | + | 1/400 |
| 5 | + | – | – | A | A | A | – | A | + | + | 1/400 |
| 6 | – | A | A | A | – | A | A | A | ± | + | 1/400 |
| 7 | – | A | – | A | A | A | A | A | ± | – | – |
| 8 | + | A | A | A | A | A | A | A | ± | + | – |
| 9 | + | A | – | A | – | A | – | A | + | + | – |
| 10 | + | A | – | A | – | A | A | A | + | + | – |

The organism most frequently recovered (Number 1) was a small Gram-negative motile cocco-bacillus somewhat resembling *Bacillus proteus*.

On nutrient agar the colonies are round, moist and white, but give no sign of spreading on this or any other media. On blood agar plates there is no hæmolysis till the second or third day, when a small zone appears round each colony. In nutrient broth a uniform growth appears in twenty-four hours and settles in two or three days. In milk there is neither clotting nor digestion. In blood serum there is no digestion—gelatine is not liquefied. The organism ferments dextrose, saccharose, maltose, galactose and lævulose, with the production of acid and gas. It produces indol, but gives no reaction to the Voges-Proskauer test. It reduces methylene blue and nitrates to nitrites, but does not produce ammonia. Cultures contain a catalase.

This organism, or a closely similar one (Number 2), which differed from it in not fermenting saccharose and in not agglutinating with a serum prepared against Number 1, was present in the mouths or wet venom, or both, of 47% of the snakes examined, but was not found in the freshly caught brown snakes, black snakes and tiger snakes.

Of the seven tiger snakes with "cancre", one or other of these two organisms was isolated from six and from six other tiger snakes which had been some time in captivity, but which had apparently normal mouths.

Of the seven copperheads with "cancre", one or other of these organisms was isolated in three only, and it was present in seven other snakes, including three which were freshly caught.

It will be observed that we have not regularly isolated from snakes with "cancre" any organism which we can regard as the probable ætiological agent.

All the organisms in Table I fail to produce acid or clot in milk. They neither digest blood serum nor liquefy gelatine. None gives the Voges-Proskauer reaction or produces ammonia. All yield a catalase and reduce methylene blue and nitrate to nitrite. None of them agglutinates with antisera against Flexner, typhoid, paratyphoid *A* or *B*, Gärtner or suipestifer. Antiserum prepared against Number 1 agglutinated all the organisms isolated which gave the same biochemical and cultural reactions as Number 1. This antiserum gave no reactions with either strain of *Bacillus proteus* which we isolated, nor with Warsau, *vulgaris*, X 19 or Kingsbury, using both H and O emulsions.

### *Staphylococci.*

From 63% of the snakes' mouths coccal organisms were isolated (*Staphylococcus albus* or *aureus* in 30 and streptococci in 1). In the 17 freshly caught snakes staphylococci were present in 12 (70%).

### *Bacillus Subtilis.*

The concrete floor in the cages is freshly covered with straw after every milking, and it is surprising that *Bacillus subtilis* was only twice isolated from snakes' mouths and three times from wet venoms.

### *Anaerobes.*

Two or three strains were present in mixed cultures from the mouths of all the snakes examined, and from many four or five different anaerobes were found. Among those present were *Clostridium septique*, *Clostridium welchii*, *Clostridium sporogenes*, and *Clostridium histolyticum*, but mixed cultures were rarely pathogenic to guinea-pigs. Great difficulties were met with in attempting to isolate in pure culture these and other anaerobes, and since, as will be seen, they were rarely present in the wet venoms, we did not think it worth while to investigate them further.

### *Organisms Isolated from Wet Venom.*

Of the 50 samples of wet venom investigated, 15 were sterile, 18 were contaminated with one organism, 12 with two organisms, 4 with three organisms, and 1 with four organisms. Of the total contaminating organisms, 31, including 6 *Bacillus proteus* and 1 *Bacillus pyocyaneus*, were non-lactose-fermenters, 11 were coliform, 8 were staphylococci, 5 were anaerobes, and 3 *Bacillus subtilis*.

This rather rare occurrence of anaerobes in the wet venom speaks strongly for the efficiency of the method of collection through rubber dam for the avoidance of gross contamination with saliva. Naturally, venom collecting as a drop on the tip of the snake's non-sterile fang cannot hope to escape occasional contamination.

### *The Bacterial Contamination of Dry Venom.*

Of the 50 corresponding samples of dry venom, 27 (54%) were sterile, 17 (34%) contained one organism, 4 (8%) two different organisms, and 1 (2%) three organisms. Of the single contaminants three were *Bacillus subtilis* (two being present in the corresponding wet venom), two were diphtheroids (not present in the original wet venom); six of the single and three of the mixed contaminants were cocci (four of these were not present in the wet venom); five of the single and five of the mixed contaminants were non-lactose fermenters; one single and two of the mixed contaminants were coliform organisms.

Of four venoms examined after only forty-eight hours' drying, all were contaminated with non-lactose-fermenting organisms; of eight subjected to drying for four days, four were sterile, one was contaminated with cocci (wet venom sterile), one with cocci and alkaligenes, one with a diphtheroid (wet venom sterile) and one with *Bacillus subtilis* (wet venom sterile); of twelve subjected to nine days' drying, seven were sterile, two were contaminated with *Bacillus subtilis*, one with a diphtheroid (wet venom sterile), one with a coccal organism (wet venom sterile), and one with cocci and a non-lactose-fermenter. Of nineteen dried for from ten to fourteen days, eleven were sterile, four were contaminated with cocci (in two cases not grown from the wet venom), one with cocci and coliform organisms, two with coliform organisms and non-lactose-fermenters, and one with non-lactose-fermenters alone.

It appears that the majority of the non-lactose-fermenters are killed out by drying and that a high proportion of the contaminating organisms in venom subjected to prolonged drying are air-borne or other laboratory contaminations. It is of interest that spore-bearing anaerobes were so infrequent in the dried venom.

Bacterial contamination of venom could be lessened by a rigid adherence to asepsis in carrying out the present technique of venom collection. The containers should be dry sterilized and covered with sterile rubber dam. A separate container should be provided for each snake in order to avoid repeated air-borne contamination when "milking" in the open air, since, when a snake bites, it depresses the rubber and when the fangs are withdrawn the rubber flies back and air containing dust is sucked through the puncture holes. This source of contamination could be eliminated only by carrying out the "milking" in a still atmosphere. The rubber dam must be slit open and placed in desiccators in a still room, and the operation of scraping out should be done in a dust-free chamber with a sterile instrument into sterile glassware.

#### Conclusions.

Freshly caught snakes do not possess a very numerous oral bacterial flora, but in captivity the number of organisms present in their mouths multiplies enormously.

"Cancre" is frequently associated with the presence of a strain of *Bacillus proteus* or with a small Gram-negative cocco-bacillus which is described in the present communication. We have no evidence concerning the ætiology of this disease of snakes in captivity.

The organisms present in snakes' mouths in captivity include anaerobes, non-lactose-fermenters, coliform bacilli and staphylococci. Freshly collected venom contained many fewer organisms, and about 30% of samples were sterile, those infected containing usually only one or two species of organisms. Over 50% of the samples of adequately dried venom were sterile. Drying appears to kill off many of the non-lactose-fermenters.

The present methods of collection and handling of venom, though they exclude gross contamination by saliva, do not guarantee sterile dry venom. Measures for the exclusion of laboratory contamination of the venom are described.

#### Acknowledgement.

Our thanks are due to the Director, Dr. C. H. Kellaway, for help in the presentation of our results.

#### Reference.

(1) C. H. Kellaway and F. Eleanor Williams: "The Investigation of the Toxicity and Sterility of a Commercial Preparation Containing Modified Snake Venom", THE MEDICAL JOURNAL OF AUSTRALIA, May 13, 1933, page 581.

## RADON IN THE TREATMENT OF CERVICAL CARCINOMA.[1]

By W. G. CUSCADEN, M.D., F.R.C.S. (Edinburgh),
*The Women's Hospital, Melbourne.*

THE regular issue from the Commonwealth Laboratory has made it possible to treat with radon the series of cases herein recorded. The amount of shielding used in cavitary treatment is always a matter of great importance. In treating this series of cases of cervical carcinoma, six tubes were used for each case, twelve millicuries in each tube. The capillary tube containing the radon was enclosed in a gold shield, bringing the total shielding to the equivalent of 2·5 millimetres of platinum. After the cervical canal is found, often a matter of some difficulty, the cervix can in almost all cases be dilated without any anæsthetic, on account of the softness of the parts and the small amount of dilatation required for the radon tubes, which have a small diameter. Two tubes covered with rubber were inserted into the uterus in tandem. For the vaginal applicator a circular or square distribution has obvious advantages. But the more posterior tube is very apt to damage the rectum. To obviate this difficulty I have used a special pessary of vulcanite, in which the remaining tubes lie in the four fornices (see Figures I and II). The posterior

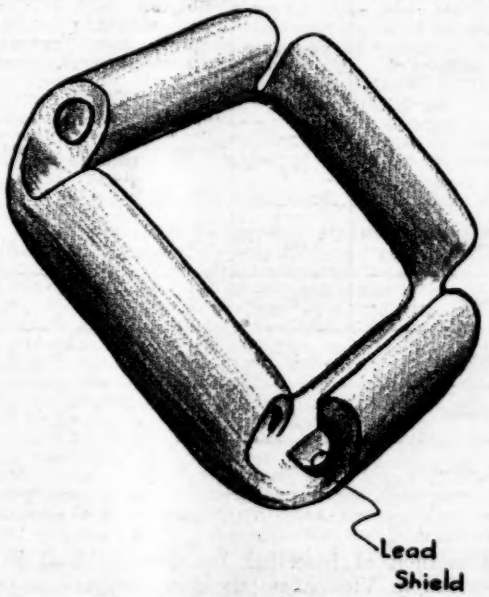


FIGURE I, showing special pessary.

tube has a lead shield, equivalent to six millimetres of lead, which protects the rectum but allows the tube to irradiate the growth. I have never had a serious case of proctitis with this arrangement. The pessary, of which there are a series of varying size, is inserted into the vagina over the spread fingers.

[1] Read at the Fifth Australian Cancer Conference, Canberra, April, 1934.

TABLE I.

*Women's Hospital, Melbourne.—Statement of Patients Treated with Radon from July 1, 1929, to December 31, 1933.*

| Carcinoma of *Cervix Uteri.* | Number of Cases Treated. | Results of Treatment. | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Apparently Cured. | | Local Improvement. | | | | Not Improved. | | | |
| | | (1) Alive and Free of Symptoms. | (2) Apparently Cured, but Died of Intercurrent Disease or Injury. | (3) Local Improvement. | (4) Local Improvement. Recurrence or Metastases. | (5) Local Improvement. Died from Metastases. | (6) Local Improvement. Died from Other Causes. | (7) Not Improved. | (8) Not Improved. Died. | (9) Palliative Relief. Died. | (10) No Information. |
| Total number of cases treated .. .. | 27 | 10 | | 3 | 2 | 1 | | | 9 | | 2 |
| Operable .. .. | 4 | 3 | | 1 | | | | | | | |
| Borderline .. .. | 8 | 4 | | | 1 | | | | 3 | | |
| Inoperable .. .. | 12 | 3 | | 2 | 1 | 1 | | | 4 | | 1 |
| Very advanced .. | 3 | | | | | | | | 2 | | 1 |

TABLE II.

*Women's Hospital, Melbourne.—Statement of Patients Treated with Radium from July 1, 1929, to December 31, 1933.*

| Carcinoma of *Cervix Uteri.* | Number of Cases Treated. | Results of Treatment. | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Apparently Cured. | | Local Improvement. | | | | Not Improved. | | | |
| | | (1) Alive and Free of Symptoms. | (2) Apparently Cured, but Died of Intercurrent Disease or Injury. | (3) Local Improvement. | (4) Local Improvement. Recurrence or Metastases. | (5) Local Improvement. Died from Metastases. | (6) Local Improvement. Died from Other Causes. | (7) Not Improved. | (8) Not Improved. Died. | (9) Palliative Relief. Died. | (10) No Information. |
| Total number of cases treated .. .. | 124 | 31 | | 6 | 12 | 4 | 4 | 9 | 51 | | 7 |
| Operable .. .. | 23 | 11 | | 1 | 3 | | | 2 | 6 | | |
| Borderline .. .. | 22 | 8 | | 2 | 2 | | 2 | 1 | 4 | | 3 |
| Inoperable .. .. | 42 | 11 | | 2 | 4 | 4 | | 3 | 15 | | 3 |
| Very advanced .. | 37 | 1 | | 1 | 3 | | 2 | 3 | 26 | | 1 |

NOTE.—20 of the above patients were treated with radon also.

The method of inserting the pessary used to be described in Vienna as "using the fingers as tram rails".

If the *levator ani* is sound, no packing is necessary to keep this pessary in place. If the levator has been torn, then gauze packing is necessary to keep it in place. The heavy weight of the lead keeps the posterior tube in place when the patient is in the recumbent position. The radon in all these cases was left in position for eight days. The shielding is heavier than is necessary, but the results are so good, both remote and immediate, in the absence of serious reaction and absence of sloughing, that I intend to continue with it, even though some $\gamma$ radiation is wasted.

It is impossible in most cases to decide whether broad ligament infiltration is inflammatory or due to extension of growth. After treatment and rest in most cases inflammatory infiltration usually disappears. Some of these patients have seemed suitable for further treatment. In these, at a later date (about two or three months), radon was implanted in the broad ligaments. The uterus was drawn well down and to one side, as in infiltrating a broad

ligament with a local anæsthetic. The introducer was then passed through the lateral fornix until it struck the uterus, and it was then slightly withdrawn; a 1·5 millicurie radon seed was left in position. By varying the angle a fan-shaped distribution is possible, five seeds being left in each broad ligament. Four seeds were inserted into the anterior and posterior fornices respectively. Eighteen seeds were used in the average case; they were left in for eight days and were removed by gentle traction on their linen threads.

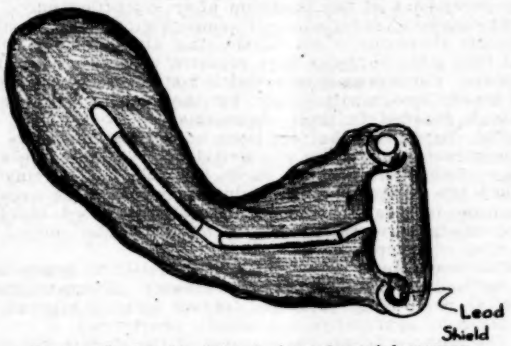


FIGURE II, showing lateral view of special pessary.

The cases in the appended tables have been classified according to the standard laid down by the League of Nations Subcommission. It is unfortunate that the condition of so many patients is so advanced when they first come for treatment. Most patients in the last two classes (inoperable and very advanced) have a history of over six months. Many have been seen by one or more medical men during this period and not examined.

## Reports of Cases.

### A CASE OF PUERPERAL INVERSION AND PROLAPSE OF THE UTERUS WITH SPONTANEOUS REPLACEMENT.

By PERCY G. BRETT, M.B., B.S. (Melbourne), F.R.A.C.S.,

*Honorary Out-Patient Gynæcological Surgeon, Women's Hospital, Melbourne.*

INVERSION of the uterus following childbirth is fortunately a rare condition, but when it does occur, often proves a tragic accident both to the patient and the attending obstetrician.

The following case is reported not only on account of its rarity, but also to demonstrate that conservative treatment may be followed by a happy result.

#### Clinical History.

Mrs. E.D., *primipara*, aged eighteen years, was rather a small, delicate-looking woman, admitted to the Women's Hospital on January 26, 1934, in a condition of marked pallor and shock and with the following history from the attendant doctor:

She had a normal confinement on January 24 and was easily delivered of a 2·75 kilogram (seven pound) child by forceps; the perineum was intact. The placenta was expelled thirty minutes later, followed by another small piece in four hours. At the conclusion of the third stage the patient was shocked out of all proportion to the blood lost, and complained of pains over the pubis.

On January 26, while the patient was straining at stool, there was a sudden prolapse of the uterus right outside the vagina. The patient was sent straight to hospital. On admission she was immediately treated for shock, being packed up with hot bottles. She was given morphine, 0·015 gramme (one-quarter of a grain). She was also given an intravenous injection of 750 cubic centimetres of 10% glucose saline solution and then a blood transfusion of 600 cubic centimetres (twenty ounces). Following this her condition improved.

One and a half hours after admission, under gas and oxygen anæsthesia, examination revealed a complete inversion and prolapse of the uterus. The uterus was œdematous and engorged, covered with blood clot and small pieces of membrane, and was grey and sloughy-looking in places. After preparing and cleansing the uterus with biniodide of mercury solution, 1 in 4,000, and wiping off blood clot and membrane, an attempt was made with hot packs and careful pressure to reduce the prolapse and to reinvert the uterus. Reinversion was found to be impracticable, but the prolapse was reduced and the vagina was packed with gauze soaked in acriflavine, 1 in 1,000, in glycerine. Twenty thousand units of gas gangrene antiserum and 30 cubic centimetres of antistreptococcal serum were administered intramuscularly and the patient was returned to bed.

The subsequent progress was as follows:

On January 27, 1934, the patient was still shocked and very pale, but her general condition had improved. The packing was changed.

On January 28, 1934, her condition was about the same. The vagina was repacked.

On January 29, 1934, she was still pale and ill. Her pulse rate was 120. Her temperature was 38·6° C. (101·6° F.) and there was a foul-smelling discharge from the vagina. Another 20,000 units of gas gangrene antiserum were administered intramuscularly.

On January 30, 1934, the packing was removed and hot potassium permanganate douches were instituted three times a day. The patient was put on an iron mixture.

On January 31, 1934, she was still very pale. A blood transfusion of 600 cubic centimetres was given from her father.

On February 1, 1934, she was better; her appetite was improving.

On February 5, 1934, her general condition was much improved; her temperature was 37·2° C. (99° F.), her pulse rate was 90 to 100. The uterus was involuting.

On February 16, 1934, the improvement was being maintained. The uterus was the size of a duck egg. The temperature and pulse had been normal for one week.

On February 24, 1934, the uterus was well involuted and lying in the vagina; there was practically no discharge. The patient was sent home with a tonic and instructions to continue permanganate douches daily; she was to return in two weeks for operation to replace the uterus.

On March 21, 1934, she was readmitted to hospital. Her general condition was excellent. Vaginal examination revealed a lax (three fingers) vagina, a small rectocele and cystocele, and a uterus which had spontaneously reinverted. The fundus was anteverted, practically normal in size, and the cervix was soft, with the external and internal os just admitting a finger. A hystero-salpingogram demonstrated a uterus of normal size and the tubes patent to their fimbriated ends.

#### Comment.

At the time of the patient's first admission to hospital more radical measures, such as Spinelli's operation and vaginal hysterectomy, were considered, but, in view of the grave risk of further shock, hæmorrhage and sepsis, were not further entertained. Even in the absence of the spontaneous reinversion which later took place, we feel that her immediate response and progress under conservative measures fully justified the treatment.

The conclusions borne out by this one case conform with those arrived at by D. N. Barrows, who reports five

cases of puerperal inversion in the *American Journal of Obstetrics and Gynecology* of January, 1934 (page 105). Three of his conclusions I quote in full:

1. The prompt treatment of shock is much more important than attempting to replace the uterus when the patient is in a doubtful condition. We can go so far as to advise packing to stop hæmorrhage before removing the adherent placenta.
2. We think of the acute case in terms of shock and not in terms of inversion of uterus.
3. Immediate replacement of the puerperal uterus after inversion is frequently attended by considerable shock and loss of blood and is a dangerous procedure.

**Acknowledgements.**

I am indebted to Dr. A. M. Hill, medical superintendent, and to Dr. J. G. Bonnin, resident surgeon, for the attention they gave to this patient.

# Reviews.

## SURGERY AND THE SYMPATHETIC NERVOUS SYSTEM.

THE appearance of "The Surgery of the Sympathetic Nervous System", by Gask and Ross, bearing the imprimatur of London, will be received with interest by many medical men in Australia. The sober and restrained account of the new surgical procedures will appeal to the conservative minded. The anatomy of the sympathetic system is briefly described and the newer knowledge of this system based on the recent surgical experiments is carefully sifted and critically discussed.

A brief history of the development of the surgery of the sympathetic system includes the researches of such well known men as Adson and Brown, and other overseas workers. While due recognition of this work is proper in such a book, it is to be noted that several important papers by Australians seem to have been overlooked. The work of Royle and Hunter is mentioned in passing, but rather too briefly when one considers that the book in question is largly devoted to what are referred to as "by products" of the Sydney work. The physiology of the sympathetic system is reviewed in some detail, and special mention is made of Penfield's case of tumour of the third ventricle producing diencephalic epilepsy and of the experimental work of Beattie, Brow, and Long, which seems to localize the cranial centre for the sympathetic in the hypothalamic region. The importance of accepting with caution the results of animal experiments is admitted by the authors, who stress the wisdom of reserving judgement on the surgical experiments in human subjects until sufficient time has elapsed for a true estimate of their effects.

An excellent account of the methods of differentiating spasmodic and obliterative vascular disease includes the work of Lewis and that of Brown.

Periarterial neurectomy is, in the opinion of the authors, of definite value in a selected group of cases such as senile and diabetic gangrene, and intermittent claudication for non-vascular ulcers of the leg. Although it is admitted that the anatomical and physiological bases for this operation are ill defined, the clinical results, both in animals and man, are sufficiently marked to justify this simple procedure in appropriate cases. Ganglionectomy replaces sympathetic ramisection as a method of dividing the vasoconstrictor fibres. For the removal of the sympathetic ganglia to an upper extremity, the posterior approach of Adson is criticized because of the removal of ribs on each side, and on account of the vascular structures which are traversed in the operation. The anterior approach is favoured. Two surgical approaches to the lumber sympathetics are described, the anterior transperitoneal, and the posterior approach as first described by Royle. The advantages of the anterior approach are obvious. Both sympathetic trunks may be removed through the one incision. The lumbar approach of Royle, however, seems to us to offer distinctly less risk of damage to colic vessels and viscera.

Some interesting observations are recorded in cases that have temporarily benefited by sympathectomy, the skin temperature showing a distinct rise, but six months later the operated limb being colder than the unoperated limb. It would appear that the work of Lewis, which suggests that both vaso-constrictors and vaso-dilators are present in the sympathetic nerve, is being confirmed by some of these clinical cases. The blood vessels develop a tone of their own, and in Raynaud's disease, especially, the direct action of cold on the blood vessels may be responsible for the recurrence of the condition after sympathectomy.

The authors' experiences of sympathectomy in *thromboangiitis obliterans* is not encouraging, although they point out that other surgeons have reported good results in this disease. Certain cases of arthritis and scleroderma appear to benefit by sympathectomy, but the authors are hopeful though guarded in their statements.

The chapter on the treatment of visceral disorders by sympathectomy is especially well done. The researches of Learmonth, Rankin, Wade, Royle, Trumble, and Barrington Ward are reviewed and a plain statement of the present position is given. Hirschsprung's disease, cord bladder, hydroureter have been definitely brought under control by section of sympathetic nerves.

The vexed question of pain and its relief by sympathectomy is dealt with in the final chapter. Dysmenorrhœa, vesical pain, renal pain, and *angina pectoris* may all be relieved by appropriate sympathetic neurectomy.

The authors favour the removal of the stellate ganglion for angina, rather than the complicated operation of Danielopulu. Doubtless the advisability of performing such operation for the removal of the danger signals of disease will be a matter of contention between physicians and surgeons, but in the present limited state of our knowledge any measure which will alleviate human suffering must be seriously considered. Causalgia is discussed in great detail and the vascular elements in this disorder are attributed to sympathetic over-activity. Periarterial neurectomy is advised as a preliminary, even curing some patients. Sympathetic ganglionectomy is the last resort in cases which fail to respond to the minor measures.

The concluding remarks of the authors remind us that "temperament" is an important factor in all disorders of the sympathetic nervous system and it is in this realm that the psychologist and the surgeon join hands. It is possible that these disorders may be treated at some future time by appropriate mental hygienic measures, and that the crude carpentry of the peripheral portion of the system be rarely necessary.

[1] "The Surgery of the Sympathetic Nervous System", by G. E. Gask, F.R.C.S., and J. P. Ross, M.S., F.R.C.S.; 1934. London: Baillière, Tindall and Cox. Crown 4to., pp. 175, with illustrations. Price: 16s. net.

# Notes on Books, Current Journals and New Appliances.

## ARTICLES ON TREATMENT.

A SERIES of articles on modern treatment in general practice has been published in *The Medical Press and Circular*. These articles achieved such popularity that they have been republished in book form.[1] Each article occupies a chapter and there are fifty-six chapters. The range of subjects is wide and the subjects chosen are such as will appeal to the general practitioner. The only article that seems inappropriate in this volume is one on modern methods of exposing the brain and meninges. Lambert Rogers describes herein the use of an electrically driven skull plough. General practitioners may certainly have to trephine a skull, but very few will possess a skull plough. However, this does not detract in any way from the practical value of the book.

[1] "Modern Treatment in General Practice", edited by C. P. G. Wakeley, D.Sc., F.R.C.S., F.R.S.E.; 1934. London: The Medical Press and Circular (Baillière, Tindall and Cox); Australia: Angus and Robertson. Demy 8vo., pp. 434, with illustrations. Price: 16s. net.

The Medical Journal of Australia

SATURDAY, AUGUST 11, 1934.

*All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.*

*References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.*

*Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.*

## SIMPLICITY.

In the character of man simplicity is a trait to be admired. The man of simplicity is open and guileless, he wears no veneer of omniscience or of impressive silence to cover his ignorance; he is direct in speech and his words are probably few; his actions are no more complex than the occasion demands. Simplicity has been sung by poets and extolled by writers of prose. Schopenhauer proclaimed it as a mark of genius, to Hazlitt simplicity of character was the natural result of profound thought, Tennyson wrote of sublime simplicity that was inherent only in the greatest men. To say that a man is simple may imply that he is mentally deficient. Simple Simon who met the Pieman would not be looked on as overburdened with intelligence, though some might suspect him of shrewdness. Simplicity of this kind need not detain us. Simplicity may be ascribed to the unsophisticated; their thoughts, their words, their mode of life are simple because they know of nothing more complex. They may be wholly admirable persons, dull perhaps, but dependable, satisfied, and on that account probably cheerful. But the simplicity acclaimed by Schopenhauer and others is different. It follows on sophistication and is bred by depth of knowledge, understanding and a sense of values. Not that all knowledgeable people are capable of simplicity; one has only to read the books of certain well known teachers of philosophy to be convinced of this. Simplicity, of course, may not always be either possible or desirable; and this brings us to the question as to whether an intelligent person not naturally given to simplicity can by some means or other acquire it. Beyond stating that in certain circumstances this may be possible, we shall not pursue the subject further, but would consider simplicity as it concerns diagnosis and treatment in medicine.

The medical student, on his introduction to clinical medicine, is taught to use his bodily senses—he learns to look, to touch, and to listen, sometimes to smell and even to taste. These simple processes are, of course, the basis of all clinical examination. Whether sufficient emphasis is laid on them depends on the tutor, surgical or medical—on the mental calibre or, shall we say, on the mental outlook of the tutor. All too soon the addenda of clinical examination, the invaluable aids of the laboratory, are introduced. Here at once is something that the student can see—the reaction in the test tube, the appearances under the microscope, the shadow on the skiagram. What need is there, so it appears to him, to bother further? The student is becoming sophisticated and is passing on to greater knowledge; but he has as yet no sense of values. If he does not acquire a sense of proportion he will use what appears to him to be the easier road, the road of the laboratory, and will forsake what we may call the road of the senses. Again, when the evidence provided by simple means is quite conclusive, he will want to carry out what are then unnecessary investigations. To quote an example: When a patient has every clinical evidence of bronchiectasis it is not necessary to inject lipiodol into the bronchi to make a diagnosis. This injection may be necessary to determine the situation or extent of the trouble, or may be used for purposes of treatment, but the man who uses it unnecessarily in diagnosis ceases to practise medicine as an art. Students are not alone in having no sense of proportion. It sometimes

happens that patients referred to clinical laboratories for examination are subjected to tests of almost every conceivable kind. Again, for example, we hear of suggestions made apparently in all seriousness that what is known as hysterosalpingography (horrible word!) may be used to determine the position of the uterus, when the intelligent use of a uterine sound will at once give the information required. The patient, it would seem, must be impressed (incidentally he has to pay); and there are many patients like Naaman of old, who went away in disgust when he was told merely to wash in the Jordan and be clean—he thought that Elisha, the prophet, would surely come out to him and stand and call on the name of his God and strike his hand over the place and recover the leper. The man who panders to the Naamans of the present day is prostituting his art. Much the same may be written about simplicity in treatment, particularly about surgical operation, often so aptly called surgical interference; but why labour the point?

Simplicity, as we have seen, is a quality inherent in the character of some people. It is possessed by many medical practitioners; and they belong to the erudite, with knowledge, understanding and a sense of values. The statement has been made that in certain circumstances it may be acquired. The effort to acquire it is worth making. The first essential in the process of acquisition is the elimination of self-seeking and a devotion to the art of medicine and to the welfare of the patient. Of medical practitioners who have erudition and wisdom we may say in paraphrase, as has been said elsewhere: "By the simplicity of their methods shall ye know them."

## Current Comment.

### OLIGURIA AND ALBUMINURIA IN BRIGHT'S DISEASE.

OLIGURIA and albuminuria, classical features of failing kidneys, are of everyday significance and value, yet no satisfactory explanation has been offered as to the exact mechanism of their production. It has long been known that in many cases of subacute nephritis with obstinate œdema and oliguria the most obvious kidney changes, which are degenerative, affect the tubular epithelium, while the glomeruli seem little affected. The causes of the fluid retention have been sought elsewhere, since the glomeruli are accepted as the eliminators of fluid. From this difficulty there arose the conception of nephrosis as a metabolic disorder with secondary degeneration of the renal tubules, clinically and pathologically separable from subacute glomerulonephritis. As a result, the tissues generally were supposed to increase their water-holding qualities, and thus leave less fluid for the kidneys to excrete. It has subsequently become plain that a clear-cut separation of nephrosis from nephritis is impossible. No single feature or group of features can distinguish during life the most genuine "nephrosis" from a disease which is proved, on histological grounds, to be subacute glomerulonephritis.

The involvement of the glomeruli in nephrosis is definite, but of insufficient magnitude to explain the great degree of water retention seen clinically. It is otherwise with subacute nephritis itself, but there probably exists some common explanatory factor other than the degree of glomerular change. Professor Shaw Dunn,[1] of Glasgow, has recently interested himself in these problems and has presented a reasonable explanation of the retention of fluid in renal disease, along the lines of Cushny's well known and widely accepted theory of renal excretion. He thinks that oliguria probably depends on excessive reabsorption by the renal tubules of a glomerular filtrate which might originally be of normal amount. Any such elucidation must show how sodium chloride is retained along with the water in nephrosis, while urea is concentrated in normal degree. According to Cushny's theory, even up to 99% of the volume of the protein-free filtrate from the tufts is reabsorbed in the tubules. Factors tending towards this reabsorption are: (*a*) the raised osmotic pressure in the post-glomerular capillary which connects with the tubular epithelium, the result of concentration by loss of filtrate; and (*b*) possible selective vital activity of the renal cells. The factor acting in the opposite direction is chiefly the undeniable vital barrier to the passage of urea from tubule to blood stream. Injury by oxalates and other poisons to the cells of the first convoluted tubule results in the retention of urea in the blood. Since urea is then held in the lumina of the tubules, it cannot but exert its physical osmotic property by holding back water from reabsorption through the cells into the blood. This is the essential manner in which urea acts as a diuretic. Shaw Dunn hastens to add that he does not suggest these to be the only factors in the normal regulation of urinary volume, but that they probably play a large part. In glomerular disease this relationship is disturbed in that much water and salt are reabsorbed, without retention of urea, that is, glomerular filtration is undiminished. The alternating actions of the glomeruli, in that one will work only for a short period and then close

[1] *The Lancet*, May 26, 1934.

down for a time, have been observed in the frog and are assumed by Shaw Dunn to occur in man. Given a constant blood flow to three adjacent nephrons (glomeruli and associated tubules), one may be assumed to be open, the others closed. The opposing factors of the osmotic properties of urea in the open lumen and of the blood in the adjacent tubules are such as produce a normal urine. If all three tubules are open together, the osmotic pressure of the urea per unit area is reduced to one-third, while that of the blood remains the same. This should result in an oliguria, but with normal urea concentration. Damage to the glomerular tufts with swelling of the endothelium would interfere with their closure and with the alternation mentioned above. The obstruction of blood flow itself is compensated for by a raised blood pressure in subacute nephritis, but hardly in nephrosis, where the changes are so much slighter. Staining of the capillary basement membranes by Mallory's connective tissue stain, however, shows that there is in nephrosis a fixed patency of the arterioles in the tufts, differing strikingly from the varying degrees of collapse and dilatation seen in the normal kidney. This probably means a permanent uniform distribution of the available blood and interferes with the normal rhythmical contractility of the capillaries, providing a histological basis for the theory of over-reabsorption of glomerular filtrate.

Shaw Dunn next discusses the cause of the heavy protein loss in the urine in nephrosis. The view that this protein differs from ordinary serum protein has been abandoned. Its origin is supposed to depend on an increased permeability of the capillary walls of the glomeruli. The actual concentration of the protein in the glomerular filtrate is really quite low, no more than is found in any transudate elsewhere, but the high albumin percentage in the urine is the result of tremendous concentration in the tubules by water resorption. The permanent state of slight dilatation already described leads to stasis and consequent greater permeability. Shaw Dunn claims then that the high percentage of albumin is due to mechanical principles of reabsorption, its large daily amount to a greater amount of blood passing the tufts. He has not forgotten the well known tubular changes and their possible contribution to these processes. He dismisses any such likelihood on the grounds of the ability of the tubules to reject the absorption of urea as good evidence of their vitality, no matter how degenerate the microscopic appearances may be. He states that it is not unreasonable to suppose that the features of other forms of nephritis shared by nephrosis are also explicable along similar lines; amyloid disease in particular is primarily a glomerular disturbance and frequently presents a clinical picture indistinguishable from nephrosis.

Thus we have a fascinating example of the modern trend in medicine and pathology, namely, to consider both from an essentially vital viewpoint. Moreover, if subsequent work proves Shaw Dunn to be correct in his assumptions, an example is given of how an astute pathologist can make a valuable contribution to a still unsettled but fundamentally important physiological problem, that of renal secretion. Cushny's theory has stood the test of much experiment and criticism during the past eight years. It seems to explain these anomalous features of nephrosis better than any other hypothesis. As Shaw Dunn remarks: "It is a reasonable presumption in pathology, that in disease an organ performs its function in a manner as nearly normal as the interfering factors will allow, and does not proceed to act upon some entirely different principle."

## HÆMATEMESIS IN PEPTIC ULCER.

IN April, 1933, the mortality from peptic ulcer was discussed in these pages in the light of reports from the General Hospital, Birmingham, and Saint Thomas's Hospital, London. The mortality at Birmingham was 13·3% and at Saint Thomas's Hospital 27%. G. Burger and S. J. Hartfall have reviewed the cases occurring at Guy's Hospital during the period 1921 to 1930 inclusive.[1] During this period 101,055 patients were admitted to the medical and surgical wards. Of 2,145 peptic ulcers 888 were gastric, 774 were duodenal, and 34 gastrojejunal. Hæmatemesis occurred in 177 cases, and in 137 of these (77·4%) the hæmorrhage was due to peptic ulcer. Of the 137 patients, 31 died (22·6%); the 31 represent 1·21% of the total ulcer cases. If 13 fatal cases in which surgical operation was performed are excluded, the percentage mortality is 15% instead of 22·6%. In our previous discussion of this subject special reference was made to surgical operation. It is therefore interesting to note that of the 137 patients 20 were operated on. In five cases bleeding followed operation for ulcer and all five patients died. Fifteen patients were operated on at varying intervals after hæmatemesis occurred. Details of these cases are given. Two patients operated on within ten days of the occurrence of hæmorrhage, died. Though the numbers on which the conclusion is based are small, Burger and Hartfall appear to be on sound ground in stating that "only rarely and in very specially selected cases can surgical intervention ever be justified".

Burger and Hartfall found that the most serious prognostic sign was the recurrence of hæmatemesis within a few days. Patients who had two or three recurrences had a three times, and patients with four or more recurrences had an eight times less favourable chance of recovery compared with those patients who had a single hæmatemesis. When recurrent hæmatemesis followed at short intervals the prognosis was better than when the recurrences were separated by longer intervals of time. The quantity of blood vomited in a single hæmatemesis and the lowering of hæmoglobin value did not form a reliable prognostic guide.

[1] *Guy's Hospital Reports*, April, 1934.

# Abstracts from Current Medical Literature.

## BACTERIOLOGY AND IMMUNOLOGY.

### Immunity in Typhoid Fever with Relapses.

G. D. C. THOMPSON AND E. E. ECKER (*The Journal of Infectious Diseases*, March-April, 1934) report a case of typhoid fever in which two relapses occurred and during which there was a complete absence of agglutinin response. The patient was a girl, aged thirteen, admitted to hospital on the eighth day of illness. She was acutely ill and had a high temperature, a palpable spleen, and rose spots on the abdomen and thorax. The initial leucocyte count was 5,500 per cubic millimetre, it dropped to 3,500 per cubic millimetre in six days, and gradually rose to 5,300 per cubic millimetre. The urine failed to yield any organisms of the typhoid group on four occasions and the bile yielded no typhoid bacilli on culture twice. Of fourteen stool cultures only one yielded *Bacillus typhosus*, and of eleven blood cultures two yielded *Bacillus typhosus*. The first positive blood culture was obtained on the eighty-seventh day of the illness and the second was accidentally grown from a specimen of blood collected for a Widal test on the ninety-fifth day. A series of twenty agglutination tests, microscopic and macroscopic, with three different types of *Bacillus typhosus*, all gave no reaction; the same result followed agglutination tests with *Bacillus paratyphosus A* and *B*. By the twenty-ninth day the first attack subsided and the temperature appeared normal. The second attack began on the fifty-third day and subsided on the sixty-fifth, and the third lasted from the eighty-fourth to the ninety-sixth day. The clinical picture was the same in all three attacks. The organism recovered from the blood and stool was a motile, Gram-negative bacillus giving the sugar reactions of *Bacillus typhosus* and agglutinated by specific anti-typhoid sera. Injected into rabbits, an "O" serum with a titre of one in twenty thousand was obtained. No agglutination or precipitin reactions were obtained with the patient's serum during the course of the disease. No bacteriophage was isolated, and the patient's serum failed to dissociate the strain of *Bacillus typhosus* isolated.

### The Isolation of Brucella Abortus from Apparently Healthy Swine.

WILLIAM H. FELDMAN AND CARL OLSON, JUNIOR (*The Journal of Infectious Diseases*, January-February, 1934), report their findings in an examination of one hundred and two head of apparently healthy swine. All were in good condition and apparently normal in every way and from seven to twelve months old. Four to five cubic centimetres of blood were collected from the coccygeal artery and serum for agglutination tests was obtained. The sera of two of the animals yielded definitely positive reactions with emulsions of *Brucella abortus*, and these animals were subjected to detailed *post mortem* examination. In one the only gross abnormality detected was an encapsuled abscess, approximately two centimetres in diameter, involving portion of the spermatic cord. Material for histological and bacteriological examination included this abscess, portions of liver, spleen, two apparently healthy lymph nodes from the head and the cervical region, and five cubic centimetres of urine. Direct cultures from an emulsion of the abscess of the spermatic cord gave negative results. Two guinea-pigs were injected with portions of each of the tissues secured at autopsy. In the second case no gross evidence of disease was discovered *post mortem*, but portions of liver, spleen, lymph nodes, diaphragmatic muscle, and a specimen of urine were again all injected into two guinea-pigs each. The guinea-pigs were all examined when they died or were killed at the end of eight or nine weeks. Cultures from the spleen were made in each case. In the group of guinea-pigs injected with tissue from the first case *Brucella abortus* was recovered in splenic cultures from those animals which received material from the spermatic abscess and one of the lymph nodes. In the group of guinea-pigs injected with material from the second case *Brucella abortus* was recovered in splenic culture from one of the animals which received material from the spleen. The organism recovered in each instance was identified as *Brucella abortus* by virtue of morphology and agglutinability by specific rabbit sera. The sera of three of the guinea-pigs also agglutinated an emulsion of *Brucella abortus* in a dilution of one in eight hundred. These observations are taken to indicate that bacteria of the Brucella group may exist in the tissues of apparently normal swine without giving rise to discernible symptoms of the disease.

### The Carrier Problem in Meningococcus Infections.

GEOFFRY RAKE (*Journal of Experimental Medicine*, May, 1934) studied the carrier problem in meningococcus infections. Three groups of normal persons considered as non-contact groups were investigated. Straight unprotected swabs and those on curved wires enclosed in protecting West tubes were used in the collection of the material, and care was taken that only material from behind the soft palate was examined. With experience, as good results were obtained with the straight tube as with the West tube. Great care was taken to protect the swab from being contaminated with saliva, as it was found, in corroboration of Gordon's work, that such contamination inhibited the growth of the meningococcus. Rabbit's blood agar was the medium used. The first group was composed of twenty-four persons working on one floor of a building unit of the Rockefeller Institute in New York City. Only three had much laboratory contact with strains of meningococci, owing to the nature of their work. Ten of these individuals were found to carry meningococci in their naso-pharynx at some time during the twenty months they were under observation; five were constant carriers, two were intermittent, and three were transient carriers. Only one of these carriers came in contact with meningococcus research work in any way. Two non-carriers handled meningococcus cultures constantly and another two handled such material occasionally. These four remained free throughout the whole period, although one twice received accidentally about two cubic centimetres of living meningococcus broth culture into his mouth. Four of the five were chronic carriers for periods ranging from twenty-one to twenty-six months; the type of organism was always the same, and in the single case, in which there was apparently freedom from carrying for a period, this corresponded with the appearance in the naso-pharynx of another pathogen. In neither of the intermittent carriers did the strain remain the same throughout the investigation. It was noted that pharyngitis or any infection associated with one of the pathogenic organisms in large numbers caused the meningococci to decrease. The second group investigated consisted of twenty-five young girls and female infants from fourteen years to six months. These were all strictly isolated in a separate ward of a foundling hospital. Among the twenty-five, two were found to harbour meningococci. The third group comprised five hundred and sixty-nine young men between the ages of eighteen and twenty-five years in a concentration camp. Each was examined on only a few occasions. Twelve carriers were found. The results obtained show that no relief from the carrier condition can be based on three consecutive normal swabs at weekly intervals, since apparent spontaneous cures, as evidenced by normal swabs, may last for four and a half months. The viability of the carrier strains, when they are sown in defibrinated rabbit's blood, is low compared with typical and freshly isolated strains of meningococci, but the exact significance of this fact is not known.

### Media for the Cultivation of Corynebacterium Diphtheriæ.

ELLEN KINBERLY AND MARGARET BEATTIE (*Journal of Infectious Diseases*, March-April, 1934) compared Löffler's medium and a medium recommended by Wahby for the growth of diphtheria bacilli. The latter medium is composed of equal parts of beef infusion, dextrose-maltose agar and

sheep plasma which has been treated with sodium albuminate. Cystine was added to the stock medium before each portion was tubed. Of 344 inoculations examined, 85 showed diphtheria-like organisms on both media, and five showed them on Löffler's medium only. A quantitative comparison was made in 39 instances by counting the diphtheria-like organisms in ten fields of stained preparations made from the growth on both media. Smears from the growth on Löffler's medium averaged ten or more organisms per field in 28 instances, while smears from growth on Wahby's medium averaged only one to four organisms in 26 instances. The authors also note that the appearance of *Corynebacteria diphtheriæ* grown on Wahby's medium was confusing. Deeply staining granules were rarely present after eighteen hours' incubation, and in many instances the bacilli closely resembled *Corynebacterium hoffmani*. Subcultures of these doubtful organisms on Löffler's medium often yielded typical *Corynebacteria diphtheriæ*.

## HYGIENE.

### A Comparative Study of Susceptibility to Diphtheria in the White and Negro Races.

J. B. BLACK (*American Journal of Hygiene*, May, 1934) has made an analysis of the mortality from diphtheria during the ten-year period 1915-1924 in ten southern states and seven southern cities, together with New York. He found a distinctly lower mortality per 100,000 in negroes as compared with whites, and even after standard age adjustment the whites still gave higher figures. In states the adjusted annual death rate in negroes was only 42% to 72% of the white rate. Memphis and New York cities alone showed a definitely worse figure for coloured people. A study at each age of the specific death rate reveals the fact that the negro rates in the first year of life are consistently higher in the states, even up to double that of white death rates. The white rate is excessive in the periods one to four and five to nine. In adult ages (especially in cities) the negroes again supply an excess of deaths, and this behaviour holds good even in certain cities where the total negro mortality from diphtheria is higher than that of the white. More extensive exposure to diphtheria in the first year of life might explain the higher figures for the first twelve months and lower in the succeeding nine years, but the Schick tests do not reveal any more general immunity at school age in negro children, indeed rather the reverse, and this, together with the heavier mortality in the adult negro, suggests a racial difference in reaction. At Baltimore the later susceptibility does not show in the mortality rates, though the first year excess and later freedom in the ages one to ten is again clearly indicated. No significant difference was considered to exist in the diphtheria carrier rates as found by Doull and Fales. In a small group of children tested for antitoxin content at the end of sixteen to thirty days after the last dose of the immunizing agent the coloured children showed a somewhat higher response. Conclusive evidence of racial difference, if any, will require more extensive testing.

### The Isolation Time of Scarlet Fever.

GORDON AND BADGER (*American Journal of Public Health*, May, 1934), discussing the isolation time of scarlet fever, state that the measure of efficiency is taken as the infecting case rate, namely, the percentage of patients responsible for familial infection within thirty days after release. ("Negative" cultures before release were required in all patients with suppurative complications.) Age of patient, presence of complications and season influenced the incidence of secondary cases. Children under five gave an infecting case rate of 5·5, eleven times greater than for all ages over fourteen. The summer rate was less than that for winter or autumn (July-September, lowest; October-December, highest). After actual trial the authors claim that shorter isolation periods are justified. The infecting case rate did not increase when adults were isolated for periods as short as two weeks and children for three weeks, providing the infection was uncomplicated. The younger group are restricted to the home premises for an additional week of observation. Adult patients released after fourteen days convalesced satisfactorily and avoided further loss of employment time. An investigation was made of 1,065 persons with reported scarlet fever, together with family, school and neighbour contacts, especially playmates. Of these, 402 were isolated in hospital, 663 at home. Apart from recognized cases, 253 (23%) additional persons had acute fever, sore throat with eruption or desquamation, or both, yet were not reported or isolated. Missed cases and atypical cases probably amount to even more than this. Familial contacts (apart from definite scarlet fever cases) were observed during and immediately after isolation. Sore throat occurred in 180, 28 had fever, and 19 vomited. Complications, such as *otitis media*, suppurative and catarrhal rhinitis and sinusitis, and cervical lymphadenitis, occurred in similar proportion in these suggestive infections, as in scarlet fever itself. "Common colds" occurred in 158 with similar frequency of complications. The authors therefore estimate that, in addition to 1,065 reported cases, 253 (22%) definite and 338 (31%) possible sources of infection existed, a total of 1,656 in all (55% additional). This probably explains why rigorous control in reported cases shows no marked reduction in the incidence of the disease. In 1,833 cases, 93% of patients developed the rash on or before the third day of onset and 99% before the end of the fourth day. Isolation after the seventh day is of little influence to prevent secondary cases. From 1,773 cases 395 secondary cases occurred; 42% of these occurred before isolation began and another 24% occurred before completion of the incubation limits. In not less than three-fifths of all secondary cases does infection take place before control is established. More secondary cases occurred during isolation under home conditions, but this was largely made up for by the greater number of secondary cases following the patients' release from hospital (total: 3·5% hospital, 3·8% home). While in 71% of home cases the patients were released in minimum time, only 46% of hospital patients were sent home; a week later the percentages were 95 and 73 respectively. More rigid observation and more severe cases may explain the observation, but the possibility of hospitalization with reinfections with other types of streptococci still needs attention. The authors consider that in scarlet fever about one-fourth of all patients really need isolation in hospital and that more rigorous control of home isolation for the first week is needed.

### Cataphoretic Velocity and Virulence of Streptococci.

EDWARD C. ROSENOW (*American Journal of Hygiene*, January, 1934) has investigated the cataphoretic velocity and virulence of streptococci isolated from throats of human beings, from raw milk, flies, water, sewage, and air during epidemics of the common autumnal cold. A special medium was used, 0·2% dextrose meat infusion broth to which three cubic centimetres of brain substance was added to twelve cubic centimetres to give pH 7 before sterilization. Ordinary media failed to give the cataphoretic velocity. A suspension of the cultures in distilled water is placed in a special cell and the time taken for twenty streptococci to traverse a unit distance (two squares or 50 microns) is noted. A direct current of 120 volts and a temperature of 24° C. were maintained. The velocity in microns per second, volt per centimetre were estimated. The naso-pharynx in colds gave a predominance of green-producing streptococci (pneumococci) (85%). The flora of the mouths of normal persons and of those with colds were very similar. Hæmolytic streptococci accompanied green-producers in only 9%. *Bacillus influenzæ* and *Bacillus mucosus* were found in 2%. The rhinotropic time and velocity, 2·5 seconds, 2·66 microns per second, volt per centimetre, prevailed early in the epidemic. During health similar organisms from the same throats in non-epidemic times had very different velocities. Streptococci of characteristic virulence by this test were isolated during simultaneous epidemics in various cities from all the sources above mentioned, the air being obtained from a recirculating plant.

# Special Articles on Treatment.

*(Contributed by request.)*

## XXXVII.

## THE TREATMENT OF FLAT-FOOT.

THE confusion which exists in the term "flat-foot" makes it essential, before discussing treatment, to give a short classification of the commoner types of the condition.

For the purposes of this paper (i) congenital and (ii) acquired flat-foot will be considered, and the acquired condition will be dealt with under two headings, (*a*) foot-strain, or the condition in which the main arch of the foot is still present, and (*b*) advanced acquired flat-foot, in which the main arch is actually depressed or even entirely obliterated during weight-bearing.

In planning treatment it must be borne in mind that the problem is essentially a muscular one and, given sound and unhampered muscles, particularly the *tibialis posticus* and the *tibialis anticus*, flat-foot is not likely to develop.

It is when the muscles maintaining the arch of the foot fail that undue strain is thrown upon the supporting ligaments of the foot, and passive stretching under the strain of weight-bearing gives rise to the painful symptoms of early flat-foot.

### Congenital Flat-Foot.

Congenital flat-foot may be noticed within the first few years of life, and is often associated with a mild degree of knock-knee.

Such children should promptly be made to develop the inverting and adducting muscles of the foot. They are usually too young to cooperate in active exercises.

The first necessity is that they must be prevented from walking barefoot or in slippers. Firm shoes should be worn, and the heel of the shoe should be altered so that the inner side is from six to eight millimetres (one-quarter to one-third of an inch) higher than the outer side and eighteen millimetres (three-quarters of an inch) longer than the outer side; this is commonly referred to as the "crooked and elongated heel".

A small leather strip should, in severe cases, be placed on the inner side of the sole, just behind the ball of the great toe. The effect of these alterations is that the child perforce must walk with the heel of the shoe flat on the ground, and in doing so must invert the foot and thus exercise the weakened muscles with every step.

Another device for compelling the child to raise the arch in walking is the "Spitzy button". This consists of a button made of layers of stiff leather, which is placed inside the shoe at a point just lateral to the position corresponding to the tubercle of the scaphoid. The button narrows from base to apex and is almost sharp at the apex. In flinching away from the button in walking the child is forced to form an arch in its foot by voluntary muscular contraction. If this method is used, frequent supervision is necessary.

If the child is old enough, active exercises for the invertors and adductors of the foot are instituted.

Sandshoes and slippers are forbidden till the arch of the foot appears to be able to look after itself.

Unfortunately, many of these congenital conditions are not seen till the child reaches school age, and they are then sometimes very resistant to conservative treatment. If such measures fail in these cases, an operation devised by Michael Hoke for flat-foot gives excellent results, but it is not advisable to perform it till the child reaches the age of at least eight years.

The operation consists essentially of an arthrodesis of the scapho-cuneiform joint with the foot in the corrected position. It is followed by the use of arch supports and by arch-maintaining exercises.

The rigidity of this joint gives the *tibialis posticus* and *tibialis anticus* muscles much better control over the front of the foot and enables them, if the after-treatment is carried out in an expert manner, to regain their arch-maintaining function.

### Foot-Strain.

Foot-strain commonly occurs in adolescence or early adult life, and particularly in people who follow occupations involving a great deal of standing. Hospital nurses are frequently sufferers. Treatment should be instituted as soon as pain referred to the arches of the feet or tiredness in the feet after exertion is complained of. If this is done, the later stages, in which actual deformity develops, can be prevented.

If the foot is actually painful, absolute rest from weight-bearing is essential until this symptom subsides. A light removable plaster of Paris cast, to hold the foot inverted with the forefoot adducted and the arch well supported, is essential in all but the mildest cases. The cast should be removed daily and the invertor muscles of the foot exercised, muscular fatigue and pain being, however, avoided.

During this stage a casting of the foot is taken and the manufacture of an arch support is put in hand.

It is to be remembered that, apart from preventing dropping of the arch, a big function of the support is to favour the action of the muscles supporting the arch when the patient is allowed to walk again.

If the patient's shoe is unsuitable (and of this more later) a suitable shoe should be procured in readiness for the ambulatory stage of treatment.

In mild cases a support need not be fitted, but the shoe should be provided with a crooked and elongated heel.

It is a common practice to leave the acquiring of the arch support to the patient. Several types are on the market and are procurable in most general stores; but, without going into the question of their merits or demerits, I would strongly advise that if a support is ordered, it should be made by a reputable firm of appliance makers to a plaster of Paris casting of the foot. The fitting of it should be supervised by the medical attendant. An ill-fitting support can do great harm by causing atrophy of the intrinsic muscles of the foot.

A properly made support should be perfectly comfortable, should really support the arch, and should favour the action of walking on the outer border of the foot.

I have found raw rubber covered with thin leather the most useful material for supports.

Country practitioners would get better results by taking their own casts or having them taken locally and forwarding them to reputable surgical appliance-makers in the cities than by merely sending the patient to a shop to buy a ready-made support.

When the patient commences to walk, daily exercises for a fixed period, preferably in the morning, should be enjoined as the most essential part of the treatment; but the amount of exercise taken should be strictly controlled so that the patient stops short of pain or a feeling of fatigue in the legs and feet.

Walking "heel-and-toe" with the feet parallel, using a line on the floor as guide, walking with the forefoot adducted and the arch maintained, and rising on tip-toe and bringing the foot down on its outer side are very useful exercises.

The use of the altered heel or arch support should be persisted in for some months after the patient is able to do the ordinary day's work or exercise without pain or fatigue.

### Advanced Acquired Flat-Foot.

In advanced acquired flat-foot the patient has passed the stage of foot-strain and has developed deformity. The arch of the foot is definitely flattened. This flattening may be present only when the patient bears weight on the foot, or in a more advanced stage it may be present even in recumbency, and the patient may be incapable of voluntarily reforming the arch of the foot.

In the first stage the treatment is essentially the same as that for foot-strain, except that proper arch supports should be fitted in every case.

The mere tilting of the heel only is not a sufficiently reliable measure.

The second stage, where the arch has passed beyond control by the patient's will even in recumbency, requires very careful consideration before treatment is decided upon.

With absolutely flat arches some patients are quite free from all symptoms and are able to undertake quite strenuous occupations involving standing and walking. This remark applies more to the congenital than to the acquired type of flat-foot, but in many acquired cases there is at this stage the same freedom from symptoms.

Other patients complain merely of the unsightliness of the deformity. In these two types of patient no treatment at all is really necessary, but in the group complaining of unsightliness treatment may be considered if the patient is made to realize the exact object of the treatment and that the prognosis as to symptoms is usually quite good even without treatment.

In the patients with advanced flat-foot who have symptoms—usually tiredness and pain in the feet and legs after standing or walking—the greatest relief is obtained from treatment the essential feature of which is manipulation.

The object of the manipulation is, by breaking down adhesions, forcibly to remould the arch of the foot. In most cases the use of some type of wrench is necessary, and considerable force has to be used. Plaster of Paris casts are applied with the feet in the corrected position and are not disturbed for a week.

The casts are then made removable and massage and gentle exercises without weight-bearing are given daily until good control of the arch is obtained by the patient. In conditions of any marked severity weight-bearing is not allowed for at least four weeks. Meanwhile casts for arch supports are taken and proper shoes are secured.

The patient is then allowed to walk with the aid of the arch supports and to do a few weight-bearing exercises. Again, it is important to avoid fatigue and pain. The amount of exercise and walking is gradually increased until the patient is able to perform the usual daily routine of life without fatigue.

Patients belonging to this type should usually be advised to wear arch supports indefinitely.

A still more severe type of flat-foot occurs when an actual proliferative arthritis develops as a result of the constant strain of weight-bearing in people with advanced flat-foot who for economic or other reasons remain active in spite of warning symptoms.

The arthritis involves mostly the astragalo-scaphoid and astragalo-calcanean joints.

Actual operative treatment in the nature of arthrodesis of the involved joints with correction of deformity is occasionally the only method of treatment in such cases, and is indicated in young people. Many patients who reach this stage are, however, elderly, and such drastic treatment should not be undertaken in them, as the cure is liable to be worse than the disease.

### Preventive Treatment.

Adequate shoeing is the principal measure of prevention. In the male this usually presents no difficulty, but in the female it requires all the persuasive powers and perseverance that the average doctor is capable of, to induce the patient to wear anything resembling a harmless shoe.

The essential feature of such a shoe is that it should be straight along the inner side from the tip to the base of the great toe, that it should have a broad heel not more than 3·75 centimetres (one and a half inches) in height, and that it should have an adequate arch-supporting sole.

The maintenance of good general health and muscle tone by proper food and exercise and avoidance of undue fatigue would prevent a great many painful flat-feet which develop in the adolescent female who, after leaving school, takes up the position of a shop assistant. She stands all day, very often on an unyielding concrete floor, and in order to enhance her appearance wears high-heeled and misshapen shoes which force her to throw the greatest possible strain on her arch-supporting muscles in order to maintain her equilibrium. Flat-foot must develop in these girls, except in the very strongest, and the medical attendant may be able to ameliorate their lot by insisting upon the wearing of shoes which are at least not entirely harmful.

In patients who have just been allowed up after a severe illness it is quite common for painful feet to be complained of. This symptom is due almost invariably to foot-strain and should be dealt with urgently by forbidding slippers and fitting with adequate shoes or arch supports in severe cases.

A. V. MEEHAN, M.B. (Sydney), F.R.C.S. (Edinburgh), F.R.A.C.S.,
*Honorary Orthopædic Surgeon, Mater Misericordiæ Children's Hospital, Honorary Consulting Orthopædic Surgeon, Hospital for Sick Children, Brisbane.*

## British Medical Association News.

### SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Geelong on May 12, 1934. The meeting was held at the Geelong Hospital. Part of the meeting took the form of a clinical demonstration by members of the honorary medical staff of the Geelong Hospital.

#### Paralytic Ileus.

DR. KEITH ROSS read a paper entitled "Paralytic Ileus" (see page 186).

DR. VICTOR HURLEY said that paralytic ileus was a difficult subject to deal with, as there was yet no agreement as to its causation. The diagnosis also of true paralytic ileus was a difficult one, as when ileus due to inflammatory causes and obstructive causes was excluded, the number of cases of true paralytic ileus was smaller than was usually supposed. While the cause of the condition remained obscure, the best treatment must remain undecided. Dr. Hurley considered that constipation alone could not be the chief cause, as after some operations the bowel was deliberately confined for six, ten or even eighteen days; yet in paralytic ileus, as soon as the bowels did act, the danger was usually passed. There was a question of the chlorides and fluids lost by vomiting. In this aspect paralytic ileus resembled duodenal fistula, but in other respects the two conditions did not resemble each other. Harris, of Sydney, in his operation for prostatectomy, had forsaken spinal anæsthesia in favour of light gas anæsthesia, because after spinal anæsthesia he found that true paralytic ileus occurred in a certain proportion of cases.

Dr. Hurley said that he usually ordered patients who had had abdominal operations an aperient on the third day. On the second day distension could usually be relieved by rectally administered fluids or by the rectal tube. If these did not succeed, he used a small glycerine enema containing about three ounces of glycerine with three ounces of water on the evening of the second day. This procedure would often start the bowel action in the right direction. Large fomentations to the abdomen were helpful, as the late Dr. Hamilton Russell had taught. Small hypodermic doses of pituitrin and the early administration of fluid by mouth tended to decrease the chance of ileus. He considered also that it was an advantage to encourage the taking of small amounts of solid food in the form of easily digestible carbohydrate, and he still had a regard for a smart dose of calomel at the time of commencing anxiety. It was desirable at all costs to avoid reopening the abdomen during the first four or five post-operative days. Regarding the use of morphine, Dr. Hurley said that he had no fixed rules. Where indicated, he gave the patient sufficient morphine to make him comfortable. In certain cases also he was sure he had been helped by the use of the stomach tube for washing out the stomach.

DR. C. GORDON SHAW expressed his appreciation of the paper and said that he had been struck by the marked degree of collapse which occurred in patients suffering from paralytic ileus. The pulse rate might be as high as 150 beats per minute, the abdomen distended, and the patient looked as if he were dying. Surgical shock seemed to be a possible cause in some cases. In some respects there did not seem to be very much difference clinically between an over-dilated stomach and paralytic ileus. The latter condition was probably a clinical manifestation of several different conditions. This possibility explained the various types of treatment adopted and the fact that each was effective in some cases. It was far better, Dr. Shaw considered, to prevent abdominal distension after operation than to attempt to cure it when it occurred. He believed in getting the bowels open on the third or fourth day by calomel or enema, or both. Fluid intake was of importance; if the patient retained fluid, he generally got well, and the converse was also true. The administration of chlorides was also helpful. In his opinion, gas gangrene antiserum was helpful in some cases, but not all. He agreed with Dr. Hurley that washing out the stomach was life-saving in some cases, and with this help the patient's condition sometimes changed for the better within an hour. Referring to the possible danger of spinal anæsthesia in causing paralytic ileus, Dr. Shaw considered it might be mentioned that, conversely, it sometimes seemed to cause the bowels to act. After ileus had occurred, he thought that the use of repeated purges, pituitrin *et cetera* was all wrong. The administration of morphine was effective in some hands and in some cases. The method probably dated back to H. O. Thomas, who used it in big doses in some cases with success. Enemata repeated at suitable intervals were often very valuable. Some patients, particularly those suffering from general peritonitis, were in desperate condition and did not respond to any method of treatment. The treatment of paralytic ileus by ileostomy or caecostomy had been advocated, but he (Dr. Shaw) had seen these methods used and drainage achieved, but the patients had died.

Dr. ROSS, in reply, said that his own practice had been very much as Dr. Hurley and Dr. Shaw had recommended, but he did not like the use of calomel, as he had known instances in which it had seemed to do more harm than good.

---

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Melbourne Hospital on May 16, 1934. The meeting took the form of a series of clinical demonstrations by members of the honorary staff. Parts of this report have appeared in the issues of July 14 and August 4, 1934.

### Hæmochromatosis.

DR. T. A. F. HEALE showed a male patient, aged fifty-nine years, who first attended the diabetic clinic on September 29, 1932. He had developed the usual diabetic symptoms eighteen months previously. On examination the patient's appearance was striking; the normal skin pigmentation was increased, and over the forehead, face, the back of the hands and forearms, and the lower extremities the characteristic greyish-brown pigmentation of hæmochromatosis was well marked. The liver was enlarged and firm, reaching 7·5 centimetres (three inches) below the costal margin. The spleen was just palpable. The diet on admission was: carbohydrate 60 grammes, protein 68 grammes, fat 135 grammes—calories 1,727—with insulin 25 units daily. At present the patient was in moderately good health, and the diabetes was satisfactorily controlled on the above diet with 60 units of insulin daily (36 units before breakfast and 24 units before the evening meal). The fasting blood sugar was 0·15%.

Dr. Heale said that hæmochromatosis was a rare disease; there had, however, been two other cases in the diabetic clinic during the last four years. The first patient was a male, aged thirty-eight years, who was admitted as a sufferer from *diabetes mellitus*. He had a slightly enlarged liver, but no pigmentation of the skin. Two weeks after discharge from the ward he was readmitted, complaining of severe pain in the upper part of the abdomen on the right side and of twelve hours' duration. This was regarded as an attack of acute cholecystitis. Later the radiologist reported the presence of multiple gall-stones. The patient was finally operated upon; the gall-bladder was normal and contained no stones; the liver was enlarged and firm, and there was evidence of a fairly recent perihepatitis. A portion of the liver was removed for microscopic examination and the pathologist suggested a diagnosis of hæmochromatosis. The patient, after operation, rapidly became worse and died in coma, which was more of the hepatic type than of the diabetic type. Autopsy revealed the typical picture of hæmochromatosis. The second patient was a male, aged fifty-four years, who had been admitted to the Austin Hospital with a diagnosis of primary carcinoma of the liver. While in hospital he developed *diabetes mellitus* and was referred to the Melbourne Hospital for treatment. Examination revealed marked typical pigmentation of the skin, a liver enlarged down to the level of the umbilicus, and *diabetes mellitus*. The patient did well on diet and insulin, but recently had developed tuberculosis of the lungs.

Dr. Heale said that the combination of pigmentation of the skin, enlargement of the liver and *diabetes mellitus* was pathognomonic and the diagnosis was confirmed by the demonstration of hæmosiderin in a section of excised skin. Difficulties in diagnosis arose when only one or two of the above clinical signs were present. Hæmochromatosis should be suspected in any patient with an enlarged liver, and especially when an enlarged liver was associated with *diabetes mellitus*. The ætiology of hæmochromatosis was unknown; Mallory and others had suggested chronic copper poisoning as a factor. Pathologically the disease was characterized by the deposition of pigment throughout the body, and this produced a progressive sclerosis of various organs, notably of the liver and pancreas. The pigment was of two kinds, hæmofuscin, which was iron-free, and hæmosiderin, which contained iron. Hæmochromatosis was a progressive malady and the prognosis was unfavourable. Death resulted from hepatic failure, diabetic coma or intercurrent disease. The diabetes was usually a late development in the course of hæmochromatosis, and many patients died before they reached that stage. The diabetes became progressively more severe and finally was insulin-resistant. Joslin had reported a patient who died in diabetic coma in spite of 1,680 units of insulin administered daily.

### Acromegaly and Diabetes Mellitus.

Dr. Heale also showed a male patient, aged fifty years, who was referred to the diabetic clinic on August 29, 1933. Sugar had been found in the urine six months previously, when he complained of thirst and polyuria. On examination he furnished a typical example of acromegaly. The urine contained a large amount of sugar, but no ketone bodies. The fasting blood sugar was 0·21%, and following the administration of 50 grammes of glucose the blood sugar rose to a maximum of 0·29% at the end of two hours. X ray examination showed a greatly enlarged *sella turcica*, with marked increase in depth, but no apparent erosion of the floor. The posterior clinoid processes were thinned, but their tips were still well defined and curved forwards. Vision was normal and there were no defects in the visual fields. The basal metabolic rate was +41%.

The acromegaly was of many years' standing, a photograph taken in 1912 showing quite definite early signs. During all these years he had not been inconvenienced by this malady, and until recently he had worked at his trade as a blacksmith. The diabetes had been satisfactorily controlled on a diet of carbohydrate 154 grammes, protein 70 grammes, fat 75 grammes—calories 1,571—with insulin 28 units daily (16 units before breakfast and 12 units before the evening meal).

Dr. Heale said that an adenoma of the anterior lobe of the pituitary, composed of eosinophilic cells, was demonstrable in almost all acromegalic persons. In the rare cases in which no tumour was found, hyperplasia of the eosinophilic cells was present. Glycosuria was common

in acromegaly; in 100 cases of proved acromegaly Cushing found glycosuria in 25% and *diabetes mellitus* in 12%. The glycosuria was often transitory, disappearing and reappearing according to the activity of the pituitary, and therefore it was highly probable that the above figures would be greatly increased if acromegalic patients were observed for longer periods. The severity of the diabetes also fluctuated, though at any given time it did not differ essentially from ordinary diabetes. The exact mechanism of the glycosuria and of the diabetes was not entirely clear, but it was reasonable to suppose that the secretion of the eosinophilic cells of the pituitary interfered with the production of insulin by the pancreas. Glycosuria was distinctly uncommon with other types of pituitary tumour.

Dr. Heale did not think that operation was indicated at present in this patient. Interference with vision was the important indication for operation; so far the patient showed no signs of pressure on the chiasma. Should this occur later, operation would be advised. Eosinophilic adenomata were sensitive to X ray irradiation and good results had been reported in many cases. Such treatment was, however, not entirely free from danger. The swelling and necrosis induced in the adenoma by the irradiation sometimes initiated pressure on the chiasma or led to an increase of pressure, as shown by rapidly failing vision. In such circumstances prompt operation was necessary. Because of this possibility Dr. Heale did not advise X ray treatment in this case.

### Chromophobe Adenoma of the Pituitary Gland.

Dr. Heale showed the X ray films from a male patient, aged fifty-seven years, who had a chromophobe adenoma of the anterior lobe of the pituitary gland. Since 1918 he had complained of lack of energy and gradual loss of weight. His appearance immediately suggested pituitary insufficiency. The skin was smooth and delicate; the face and neck were finely and prematurely wrinkled; the complexion was pasty; the hair was scanty and of fine texture, and this was especially true of the axillary and pubic hair. X ray examination showed a greatly enlarged *sella turcica*. The patient had worn glasses for the past seven years and with them visual acuity was normal. The fields of vision showed a slight general contraction, equal in each eye. Operation had not been advised at present.

### Aneurysm of Thoracic Aorta.

DR. GEOFFREY A. PENINGTON demonstrated an interesting series of cases of aneurysm of the thoracic aorta. In four of the cases the affected portion was the ascending aorta, in the other the descending aorta. One of the cases was apparently the result of congenital infection. Electrocardiograms were within normal limits in each instance.

The first patient presented was E.C., a male, aged fifty-six years, who reported on August 31, 1933, with a history that for two years he had been unable to work because of dyspnœa, which was especially severe at night. He had been conscious of strenuous heart action and complained of pain in the lower thoracic region on both sides if tired. He was easily fatigued and had noticed swelling of the feet and ankles at night. He had a chancre twenty-three years ago.

The blood pressure was 150 millimetres of mercury systolic, 90 diastolic in both arms. There was traumatic keratitis of the right eye, and iridectomy had been performed. The left pupil was slightly irregular and sluggish in its reaction to light. Examination of the precordium revealed the apex beat to be in the fifth left intercostal space, 10·0 centimetres (four inches) from the mid-line. Dulness was present to four fingers' breadth in the first right intercostal space, and to three fingers' breadth in the second right intercostal space. A visible and palpable pulsation, heaving in nature, was present in the first and second right intercostal space. A poor reduplicated first sound and soft systolic bruit were heard at the apex beat, and a booming second sound at the aortic region. A loud double bruit was audible over the area of pulsation. The blood reacted to the Wassermann test, the result being "P + ± —" by the warm method, and "P + + +" by the ice-box method. Radiological examination showed a rounded projection of the beginning of the ascending aorta extending to the right, with calcification in the right wall of the aneurysm.

The patient had been treated with iodide of potash and mercury by mouth, and "Bismol" given intramuscularly. The systolic blood pressure was then 136 and the diastolic pressure 84 millimetres of mercury.

Points of interest were the great improvement in cardiac efficiency with treatment and the absence of aortic regurgitation.

The next patient was W.M., a male, aged forty-eight years, who attended on May 15, 1933, complaining of a sensation over the right lower anterior region "as though soap bubbles were bursting". Slight pain over the same area developed. There was no history of venereal disease, but his wife had died suddenly at forty-six years from cardiac disease.

The systolic blood pressure was 150 and the diastolic pressure 90 millimetres of mercury. The pupils were equal, circular, and reacted to light and accommodation. The apex beat of the heart was in the fifth left intercostal space, 9·3 centimetres (three and three-quarter inches) from the mid-line. There was a heaving pulsation of the chest to the right and anteriorly, maximum over the right lower anterior part of the thorax. Dulness extended to 10·0 centimetres (four inches) to the right of the mid-line in the fourth and fifth intercostal spaces, and upwards to the third intercostal space; one finger's breadth of right cardiac dulness was present in the first right intercostal space. A double bruit was audible all over the precordium and extending outwards into the left axilla, being maximum in intensity in the fourth right intercostal space, 5·0 centimetres (two inches) to the right of the sternum. A palpable and audible friction rub was present over the dull area, synchronous with cardiac pulsation and maximum during inspiration.

Slight dulness and diminished breath sounds at the right base were the only abnormal pulmonary signs. Radiologically there was bulging to the right of the heart shadow, thought to be the right auricle. The blood reacted to the Wassermann test.

Treatment consisted of rest in bed and a biniodide of mercury mixture for six weeks, followed by "Novarsenobenzol", 0·1 gramme. The patient was unable to tolerate arsenic and felt ill with "Bismol". Physical signs showed little, if any, alteration beyond disappearance of the friction rub. The systolic blood pressure was then 160 and the diastolic pressure 60 millimetres of mercury, and there were symptoms of increasing insufficiency.

The points of interest were the pleuritic symptoms at the onset without pain; the slight cardiac insufficiency at the onset; the low position of the aneurysm, simulating a lesion of the right auricle; and the intolerance of active antisyphilitic treatment.

The third patient was G.T., a male, aged thirty-six years, who attended the hospital on January 23, 1933, complaining of pain and a sense of constriction about the chest, at the level of the nipples, occurring in bouts, not definitely related to exertion, often occurring at rest in bed and associated with numbness in both arms as far distally as the elbows. He felt "knocked out" after an attack. There was no history of venereal disease, but he had had "growing pains" as a youth. One sister had died from paralysis, called locomotor ataxia, at twenty years, and had had temporary blindness. His other sister was alive and well. His wife and three children were alive and well and their blood did not react to the Wassermann test.

The systolic blood pressure was 140 and the diastolic pressure 54 millimetres of mercury, and there was capillary pulsation with a water hammer pulse. Distended veins were apparent over the upper part of the thorax anteriorly. There was visible pulsation in the first and second right interspace, with a sustained systolic thrust and a systolic thrill. A visible pulsation was also present in the second left interspace. The apex beat was in the fifth left interspace, 10·0 centimetres (four inches) from the mid-line, and there was dulness to the right of the sternum to two and a half fingers' breadth in the first and second right interspace. A dull area to the left of the sternum extended for two fingers' breadth in the second

left interspace. There was a double aortic bruit and a continuous bruit with systolic and diastolic accentuation at the pulmonary area.

No other evidence of a syphilitic lesion was found, except a reaction to the Wassermann test, twelve minimum hæmolytic doses of complement being fixed. X ray examination revealed a widening of the aorta, which bulged to both sides anteriorly and slightly posteriorly, probably all confined to the ascending portion. No appreciable change had occurred subsequently. Treatment comprised rest in bed for six weeks, mercury and iodide of potash taken by mouth for one month, followed by small doses of "Novarsenobenzol" (0·1 to 0·15 gramme) intravenously and "Bismol" (two cubic centimetres) intramuscularly given every week and concurrently.

Of undoubted interest were the apparently congenital infection and the improvement in the general condition of the patient with treatment.

The fourth patient shown was J.McD., a male, aged sixty-nine years, who had been admitted to the out-patient department on January 10, 1924, at the age of fifty-eight, complaining of frequent attacks of giddiness with loss of consciousness, and of two similar attacks without loss of consciousness during the preceding two years. At that time the systolic blood pressure was 145 and the diastolic pressure 80 millimetres of mercury, and the apex beat of the heart was 10·0 centimetres (four inches) from the mid-line in the sixth left intercostal space, and a slight increase of dulness in the second right interspace was noted. A systolic bruit was audible at the apex and the aortic area, but the records contained no mention of the aortic second sound. A strongly positive reaction occurred with the Wassermann test. No aural lesion was found to account for giddiness, but X ray examination showed a general dilatation of the aortic arch, especially through the ascending portion. Giddy attacks ceased with the administration of iodide of potash and mercury.

On July 1, 1925, slight tracheal tug, Corrigan pulse, and capillary pulsation were noted, and a double aortic bruit was audible. General dilatation of the arch of the aorta with aneurysmal dilatation of the ascending portion was demonstrable radiologically on December 11, 1925.

At the time of the meeting the apex beat of the heart was 12·5 centimetres (five inches) from the mid-line in the sixth left interspace. There was a visible pulsation in the second right interspace and a diastolic shock. Right cardiac dulness extended in the second right interspace to three fingers' breadth, in the third to two fingers' breadth, and in the fourth to two fingers' breadth. The systolic blood pressure was 164 and the diastolic pressure 84 millimetres of mercury. A double bruit was present all over the precordium, being maximum in the third right interspace.

Dr. Penington had tabulated the various drugs used in treatment during the ten years, commencing with iodide of potash and mercury, which were followed by "Sulfarsinol", "Trepol", "Stovarsol", "Novarsenobenzol", "Muthanol", "Bistovol", "Bismol", "Silver Salvarsan", "Quinby", and latterly a combined course of "Novarsenobenzol" and "Bismol". It was pointed out that quantitative estimation of the reaction to the Wassermann test was of value as a guide to treatment and that it was essential to continue indefinitely with active treatment once the diagnosis of aortitis could be made. In addition to intolerance of intravenous arsenical preparations earlier in the course, bismuth stomatitis had occurred after twenty cubic centimetres of "Muthanol" had been given during ten weeks. Iodide and mercury had been given by mouth at intervals throughout.

Of outstanding interest was the fact that the condition was of ten years' duration, with reasonably good cardiac efficiency, but with a slow, steady, progressive increase in the size of the aneurysm despite treatment. The only occasion on which the serum had failed to react to the Wassermann test was after the administration of "Silver Salvarsan", but a good response had occurred with "Quinby". It was also of interest to note a tolerance of "Novarsenobenzol" (0·3 gramme) after an earlier intolerance thereof.

The last case presented by Dr. Penington was that of an aneurysm of the descending aorta in a male, H.H., aged sixty-two years. The patient had attended the hospital on April 12, 1934, with a history of abdominal pain at intervals for two years, occurring usually when he was troubled by the coughing associated with frequent colds. The pain extended around both sides of the lower part of the thorax to the abdomen. He had a chancre twenty-four years ago, and had buboes opened, but no other treatment.

The pupils were slightly irregular, but reacted briskly to light. The systolic and diastolic blood pressures on the right side were 116 and 66, and on the left side 116 and 76 millimetres of mercury respectively. On the nose there was a slight diminution of sensibility to pin prick of alar distribution. There was no enlargement of the heart to the left, but one finger's breadth of right cardiac dulness was present in the second right interspace. A typical "*bruit de Taboorka*" was heard at the aortic region. Apart from a few basal crepitations, no evidence of pulmonary lesion was demonstrable. Eleven minimum hæmolytic doses of complement were fixed by the patient's serum on application of the Wassermann test. Radiologically there was no enlargement of the heart shadow, but there was an aneurysm of the descending aorta with some distortion of the arch of the aorta. Treatment had consisted of the administration of iodide of potash and mercury by mouth.

Points of interest were the practically symptomless occurrence of an aneurysm which revealed no physical signs of its presence, the typical clinical evidence of aortitis, and the absence of aortic regurgitation.

In presenting the series of cases Dr. Penington stressed the following points:

1. Full exposure of the chest and thorough inspection were essential for the diagnosis of aortic aneurysm, which might otherwise escape notice.

2. A history of syphilitic infection was valuable confirmatory evidence, and adequate treatment at the time of the infection was the only effective method of preventing the development of aortitis and aneurysm.

3. Apparently congenital infection was the explanation of one case.

4. The "*bruit de Taboorka*" was practically pathognomonic of syphilitic aortitis and might be the only evidence of syphilitic infection. If it was present, X ray examination of the chest was an essential investigation.

5. Aortic regurgitation was usually present in a case of aortic aneurysm.

6. An electrocardiogram sometimes showed no abnormality although gross cardiac insufficiency existed.

7. The atypical occurrence and distribution of anginal pain was a helpful clinical indicator of aortitis.

8. Rest was of the utmost importance in treatment, and antispecific treatment was necessary for an indefinite period.

9. Intravenous arsenical preparations were contra-indicated until a thorough course of iodide and mercury had been given, and great caution was necessary. Only very small doses were advisable until tolerance had been acquired.

10. Quantitative estimation of the reaction to the Wassermann test was of some value as a guide to the intensity of antisyphilitic treatment.

## THE SIR RICHARD STAWELL ORATION.

DR. A. E. ROWDEN WHITE, of Melbourne, has placed with the Victorian Branch of the British Medical Association the sum of £1,000, to be controlled by trustees, with the object of commemorating Sir Richard Stawell and his work and perpetuating his memory by an annual oration. Dr. White expressed a desire that the scope of the oration should be sufficiently wide to emphasize the importance of medical science, medical teaching and scientific research upon all citizens, and that special weight be attached to the study and presentation of problems possessing a clinical aspect.

The first oration will be delivered by Dr. C. Bickerton Blackburn, of Sydney, in the Medical Society Hall on Friday evening, October 12, 1934. The title of the oration will be: "The Teaching of Clinical Medicine."

The oration will be open to all medical men and their wives, to distinguished guests from other States, and to certain others who are interested in hospital and medical matters. These will be invited in due course by Dr. Gerald Weigall, President of the Victorian Branch.

The Council of the Branch greatly appreciates the action of Dr. Rowden White in thus honouring the services which Sir Richard Stawell has given to the medical profession and to the community, and it is confident that the fruits of this generous recognition of such work will become more and more valuable with the progress of years.

# Correspondence.

## REDUCED TELEPHONE CHARGES TO DOCTORS.

SIR: Because a doctor's telephone is a convenience to the public and because there is a movement in Great Britain to reduce the telephone rentals to doctors, district nurses and others to whom the telephone is mainly a means of communication from outside callers, it would not be inappropriate for the medical profession in Australia to ask the Federal Government to introduce a bill to provide cheaper telephone service to doctors.

It must be pointed out that, though the telephone is used to arrange visits and consultations, it is also used by the public to ask questions and to seek advice for which no charge is usually made by the medical man; thus the doctor has to provide at his own cost a means of cheating himself of his own lawful fees.

Telephone rental charges should be reduced by at least 50%.

If the Federal Council of the British Medical Association would take the matter up and arrange for a bill to be introduced in Parliament, it would render a service of distinct value to the medical men of Australia.

Yours, etc.,

B. W. STEVENSON.

Waverley,
New South Wales,
July 23, 1934.

## A VISUAL PHENOMENON.

SIR: If one enters a perfectly dark room with white walls and illuminates them with a coloured light, say red, the walls appear faintly red and after a time quite or nearly white.

I asked Mr. J. S. Rogers, of the Physics Department of the University, to ascertain the nature of the light waves which reach the eye in these circumstances, and he kindly sent me the following reply:

> I have examined the effect produced by a red light in a dark room with white walls. I used a direct vision spectroscope and first looked at the light directly. It gave a band in the red only—no yellow light nor any of shorter wave length. I then examined the light from the wall. This also showed the red band but, as the portion of the wall examined was successively further from the light, the intensity of the light, of course, diminished and with the decrease in intensity the red appeared to become paler although situated in the same spectral region. This paleness made the colour appear whiter and this may account for what you mentioned. The problem appears to me to be psychological rather than physical.

It is quite obvious that it is a psychological phenomenon which is sometimes described as "regression to the normal"—words which may describe the phenomenon but do not explain anything. It is just one of the many biological phenomena which cannot be explained on physical grounds.

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,
Melbourne, C.1,
August 1, 1934.

## MEDICAL MEETINGS.

SIR: After almost forty years' experience, pleasant and otherwise, of scientific meetings of medical men, I offer some observations.

Whether at a congress, a routine meeting of a Branch, a meeting of a section, the material presented is similar. Rarely one finds that there is a message to give, the disclosure of an important discovery or the detection of a dangerous error; usually well-worn paths are trod and little deviations are advised or landmarks made plainer. All these things should be done, but could we not, on the whole, make their presentation to our fellows more acceptable? Is it too much to ask that an address should be delivered and not read? After the thinking and writing necessary to preparation, surely an occasional glance at the text should suffice. Both in the original paper and in the discussion reiteration is too common; it seems that unless a listener has some fresh point to make, or criticism or opposition to offer, he would be better silent. It is tedious to hear one after another reaffirm the faith that is in us all. Free discussion is most desirable and may fittingly contain pointed personal experiences of the greatest value, but desultory happenings with no particular application are wearisome to hear.

The chairman of such meetings is unwilling to do anything likely to prevent useful discussion, and it is to the taste and good sense of all that we must look for improvement.

Yours, etc.,

BRONTE SMEATON.

Adelaide,
Undated.

## "VENTRICULIN."

SIR: We are informed that the author of the "Liver-Diet Cookery Book", D. Sewart, gives a few suggestions for preparing "Ventriculin" as a sandwich filling, in the *Nursing Times*, June 9:

1. Blended with butter and tomato ketchup.
2. Blended with anchovy sauce and butter, with or without finely chopped lettuce or mustard and cress.
3. Blended with "Marmite" and butter.
4. Blended with a little "Bovril" and butter, with the liquid portion of chutney.

"Ventriculin" may also be stirred into a cupful of warm, not hot, "Marmite" or "Bovril" or soup, preferably thick, to which a little sauce has been added. From the patient's point of view the essential thing is to avoid monotony.

So many people suffering from pernicious anæmia are taking "Ventriculin" in Australia and New Zealand, we thought perhaps this might be useful information.

Yours, etc.,

PARKE, DAVIS AND COMPANY.
(Henry I. C. Dent.)

50, Rothschild Avenue,
Rosebery,
New South Wales,
August 1, 1934.

# Obituary.

## MICHAEL O'GORMAN HUGHES.

WE regret to announce the death of Dr. Michael O'Gorman Hughes, which occurred at Sydney on August 5, 1934.

## A WARNING.

A MEDICAL PRACTITIONER in Brisbane writes that there is in Brisbane a certain individual who poses as a sick man, but who is in reality the worst type of morphine addict. He always uses a most plausible tale in the way of stating his own pains or those of his wife or those of some fictitious sufferer, for which it is necessary to obtain hypodermic tablets of morphine. Occasionally he obtains veronal under the same pretext. In addition to his morphine craving, this man is dangerous in other respects. It would be as well if all medical men in Brisbane took pains to remember this when prescribing morphine for anybody other than actual patients under medical supervision. This individual is thin, of a darkish complexion, and looks ill, and is voluble in a correct medical description of his pains.

## Diary for the Month.

AUG. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 14.—Tasmanian Branch, B.M.A.: Branch.
AUG. 15.—Western Australian Branch, B.M.A.: Branch.
AUG. 15.—Victorian Branch, B.M.A.: Clinical Meeting.
AUG. 21.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 21.—Tasmanian Branch, B.M.A.: Council.
AUG. 22.—Victorian Branch, B.M.A.: Council.
AUG. 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
AUG. 24.—Queensland Branch, B.M.A.: Council.
AUG. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 30.—South Australian Branch, B.M.A.: Branch.
AUG. 30.—New South Wales Branch, B.M.A.: Branch.
SEPT. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 4.—Tasmanian Branch, B.M.A.: Council.

## Medical Appointments.

The undermentioned appointments have been made at the Adelaide Hospital, South Australia: Dr. I. A. Hamilton (B.M.A.), Temporary Honorary Assistant Surgeon; Dr. A. C. Savage (B.M.A.), Honorary Clinical Assistant to Surgical Section; Dr. N. T. M. Wigg (B.M.A.), Honorary Anæsthetist.

• • •

The undermentioned appointments have been made at the Parkside Mental Hospital, South Australia, for a further term of three years: Dr. E. A. Matison (B.M.A.), Honorary Surgeon to Ear, Nose and Throat Department; Dr. O. M. Moulden (B.M.A.), Honorary Gynæcologist.

## Medical Appointments Vacant, etc.

**For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xx, xxi, xxii.**

BALMAIN AND DISTRICT HOSPITAL, BALMAIN, NEW SOUTH WALES: Resident Medical Officer.
CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.
MATER MISERICORDIÆ CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: Resident Medical Officer.
PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.
QUEEN VICTORIA MEMORIAL HOSPITAL, MELBOURNE, VICTORIA: Resident Medical Officers (female).
ROYAL HOSPITAL FOR WOMEN, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.
ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Senior Resident Medical Officer.
TAMWORTH DISTRICT HOSPITAL, TAMWORTH, NEW SOUTH WALES: Pathologist.
THE EASTERN SUBURBS HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.

## Medical Appointments: Important Notice.

**MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square London, W.C.1.**

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney. | Australian Natives' Association.<br>Ashfield and District United Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham United Friendly Societies' Dispensary.<br>Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney.<br>North Sydney Friendly Societies' Dispensary Limited.<br>People's Prudential Assurance Company Limited.<br>Phœnix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association, Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Brisbane Associated Friendly Societies' Medical Institute.<br>Chillagoe Hospital.<br>Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.<br>Lower Burdekin District Hospital, Ayr. |
| SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide. | Combined Friendly Societies, Clarendon and Kangarilla districts.<br>Officer of Health, District Council of Elliston.<br>All Lodge Appointments in South Australia.<br>All Contract Practice Appointments in South Australia. |
| WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia. |
| NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.